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VOLUME THE THIRD

SESSION 1909-10

PART II

EPIDEMIOLOGICAL SECTION	LARYNGOLOGICAL SECTION
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PROCEEDINGS
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VOLUME THE THIRD

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1909-10

EPIDEMIOLOGICAL SECTION



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Epidemiological Section.

October 22, 1909.

Dr. JAMES NIVEN, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

Poverty and Disease.

PART I.

THE MEASUREMENTS, OCCASIONS, AND RELATIONS OF POVERTY TO FATAL DISEASE.

THE subject which I have chosen is one of overwhelming importance, but of such complexity and difficulty that I feel sure you will extend to me your indulgence if either you do not agree with my remarks or if you do not consider them adequate to the question. There is before us an immense material amassed and digested by highly competent observers, and two great reports by the Poor Law Commissioners which mutually illuminate one the other, and which, while both of them insisting on the grievous ailments of the body politic, approach their treatment in a comprehensive spirit—one, however, in a conservative, the other in a more radical attitude. The relation of poverty to disease has, moreover, been treated by your last President, as far as phthisis is concerned, with all that mastery of statistics which he possesses. And, in confining himself to phthisis, he has not very much narrowed the field of view, as we shall see. He has, however, refrained in the main from what may be called parochial statistics, and has deduced his conclusions from the behaviour of the disease as it affects nations. There is, on a priori grounds, much force in his contention that segregation in union hospitals, and, so far as phthisis was in the past received into general hospitals, segregation in all hospitals, must have exercised a powerful influence towards the reduction of the disease.

To those who are convinced that, under certain conditions, phthisis is readily disseminated amongst the poor, the proposition appears almost incontrovertible. There are, however, disadvantages as well as advantages in this method, and it appears desirable also to appeal to the facts of individual localities so that we may see whether these also point to segregation of advanced cases of phthisis in hospitals as a dominant factor in the campaign against tuberculosis.

Dr. Newsholme has discussed the suggestion that poverty is largely instrumental in the production of phthisis and points out that the term "poverty" is wanting in clearness, and, in fact, such is the case. What we have for the most part in our minds, when we use the term "poverty," is that deficiency of means which is of sufficient force to cause appeal to the guardians of the poor for relief in money or for medical aid. This is, undoubtedly, the best means we have for gauging the amount of destitution in any district, although it is subject to several deductions. The extent to which equally poor persons, from one period to another, have sought public assistance, has depended on the widely varying policy of Government. Nevertheless, it is probable that the number of poor people relieved from one period to another has varied in the same direction as the actual amount of destitution experienced. In the same manner it is difficult to compare, from the pauper returns, the amount of destitution experienced in one place with that experienced in another, inasmuch as the numbers relieved have changed with the widely varying policies pursued by different boards of guardians.

It is not suggested that such variations are always without justification. Thus the Manchester Union, with its large contingent of persons residing in common lodging-houses, might well show a comparatively large amount of institutional treatment when compared with the Chorlton Union and North Manchester, in which poverty for the most part affects families living in houses of their own. In the case of rural pauperism, which mainly concerns itself with the aged and infirm, it is to be expected, and is perhaps desirable, under suitable safeguards, that outdoor relief should preponderate. There are, however, variations in laxity and stringency which affect the number of persons receiving poor relief in a manner not so legitimate.

In addition to this measure of poverty there is another of primary importance for our purpose—viz., the number of deaths from sickness occurring in the union hospitals. This is, in fact, the only direct measure which we possess of the incidence of sickness on the poor.

There is, however, no reason why we should not know what are the diseases for which poor persons seek and obtain medical relief. It would be comparatively easy for some officer attached to large Union hospitals to enter on a schedule of diseases, abridged from the table of causes of death attached to the reports of medical officers of health, a monthly statement of the cases of illness for which sick persons had been admitted. This would throw much light on the relation of different classes of disease to poverty in different localities, taken along with other statistics.

Where, as in Manchester and other large towns, the sick poor are largely treated in voluntary institutions, a complete picture of the illnesses of poor persons could only be obtained by the preparation of similar returns for other hospitals. In this manner a clear idea would be obtained of the incidence not only of those diseases which terminate fatally, but also of others not of an immediately fatal character, though the picture might be an incomplete one. As matters stand, an annual classification is given, without statement of age, of the illnesses for which the sick are admitted into some voluntary institutions. But no classification is available from the union hospitals.

In the absence of general statistics we have to rely on such special inquiries as may be made from time to time. This observation, however, does not apply to illnesses terminating fatally, and it is possible to obtain by classification of the death returns the fullest information regarding these.

Even in the absence of such classification it is possible to obtain some information as to such illness from the general death statistics of the district, though such information does not in some respects possess the precision of records specifically tabulated *ad hoc*.

There is, also, another manner in which the question of the relation between poverty and fatal illness may be approached for any particular district. It may be assumed that the poorest male workers of the community are labourers, and it is easy to compare the death-rate of labourers or of other classes engaged in low-grade work at definite ages with the death-rates of other workers at the same age groups.

There is, undoubtedly, a great amount of poverty which cannot be measured by pauperism, and in which there is deficiency of the means necessary for vigorous and healthy life; that is to say, there is not enough money coming in to pay for house-rent, clothing, fuel, and sufficient food. To enable us to form an accurate judgment of the total amount of such poverty it is necessary to frame some standard of means

below which a family must be regarded as in effect destitute, and to make a house-to-house inquiry through reliable agents into the circumstances and mode of living of each family in the district. Needless to say, such an inquiry is not an easy one to make, and it is still more difficult to arrive at satisfactory results. It was in this way, however, that Mr. Charles Booth and Mr. Rowntree arrived at their estimates of the poverty existing in London and York respectively. These inquiries furnish, however, precise measures of the total amount of destitution and poverty prevailing, and also bring into relief many of the causes of poverty of this degree.

In addition, however, to the destitute families investigated by Mr. Rowntree, there are large numbers of men and women living in common lodging-houses who are not, in this sense, destitute at all, who are able to earn far more than their mode of life demands, and who can satisfy their wants on one or two days of work a week; with others who earn good wages but spend them, and who congregate in large lodging-houses, where no separate life is possible and in which disease is rife. These men nearly all belong to the ranks of casual labour in its most irregular form. They never save, and when overtaken by illness are removed to the union hospitals, where they embitter the lot of the respectable poor. Nor is this class confined to common lodging-houses.

What now are the conditions bearing on liability to disease which the poorest classes suffer? If the family is unable to pay the rent of their house, as the children come, they move to another until they arrive at the poorest class of dwelling procurable, perhaps a two-roomed house, without separate closet accommodation—although the number of these in Manchester is now happily small—but, at all events, at some poorer class of house, in some narrow and dark street, or with defective ventilation in the rear, perhaps more or less damp, and, it may be, with other insanitary accompaniments. The persons with whom they associate may have been similarly pushed by pressure of disease. In this manner the poorest class tend to congregate in the older, more central, and, if the expression may be allowed, in the more diseased parts of our large towns. There is thus increased exposure to infection. Naturally, it is the persons without settled wages who are most subject to this form of pressure. It is a matter of general experience that public-houses are most numerous in the poorer parts of the towns.

As the husband is, from one or another cause, often without the amount of work necessary to pay the rent and provide food and clothing, the wife seeks work, and the children are neglected and become dirty

and ailing. The want of adequate clothing, especially in winter, prevents their due growth, and the deficiency of food, while it tends to develop great alertness in obtaining food by all available means, serves in the main to prevent both their physical and mental growth. Their deficiency in physical growth, where actual deformity is not produced, may be regarded in a measure as conservative. But even for the smaller structures which remain there is not sufficient nourishment to produce healthy tissues, nor enough blood-corpuscles manufactured to convey an adequate supply of oxygen and to remove waste matters. All the vital cells suffer, whether of the brain, lungs, heart, bones, or digestive organs. They readily fall victims to diarrhoea and other digestive troubles, to heart affections, to meningitis, and to a variety of lung affections. If they struggle through one illness, they are more easily assailed by another. Both father and mother are under frequent, if not constant, anxiety, and are underfed. They also easily fall a prey to illness, especially of an infectious character.

In the country such children are able to run about in the fields or, at any rate, in the open. Their lungs are stimulated to increased activity, and their tissue waste is oxidized. In turn the digestive organs dispose of coarse food. Nothing is more notable about the children of the poor in towns than their rejection of plain and wholesome foods, such as porridge or herrings. This is due, no doubt, to various causes: partly to ignorance or carelessness in cooking; partly, perhaps, the child has been turned against the food by bad materials; partly it may be due to undue delicacy of digestion, the result of disease; partly it is the result of early feeding with unsuitable and spicy foods. In any case, the result is that the town child, cooped up in miserable surroundings and without the opportunity of healthy play, does not develop as does the child in the country. In the town also there is marked deprivation of light, and there is reason to believe that this is a distinct factor in health. It is, however, slight in comparison with the want of healthy exercise, which must be regarded as one of the most serious deprivations to which the town child is subjected. Another has been alluded to—viz., the ignorance of the average mother in towns. There can be no doubt that the poor woman in towns, who has usually followed some calling absorbing her energies from the time she leaves school, is in a far inferior position, on the average, in regard to the thrifty use of what means she has, and in respect of the rearing of children, to the country woman, who has had much more experience of domestic management. This is another large factor in the difference between town and country.

But, in addition, it is doubtful whether the agricultural labourer, on wages varying from 12s. to 18s. a week, is in anything like so poor a condition as the casual labourer in towns. His actual earnings are, perhaps, much the same. But, supposing he earns 12s. a week, he has a house rent free; he has a patch of land on which to grow vegetables; possibly he has a potato patch in the farmer's field; his fuel is carried for him, or he gets wood for nothing; he may keep pigs and chickens. His employment is secure if he is a steady and useful man, and his children readily get employment; he and his family usually enjoy good health. Poor as he is, his poverty is riches compared with that of the casual labourer in large towns; provisions are cheaper, clothes as cheap.

Fifty years ago both men and women in towns were worn out prematurely by toil commenced at an early age, extending over long hours, and unremitting, carried on in unwholesome workshops, and left only for a still more unwholesome home amid noisome smells and infection, dark, unventilated, crowded, and dirty. To-day the conditions of labour are much lightened, the hours of work are shortened, the injurious conditions are gone or are in process of removal, wages are higher, and food is cheaper. But new forms of distress have revealed themselves. In all our large centres of industry there is far more unskilled labour than is required. Numbers of men attend at the docks and large factories waiting for the day's work which only a portion can obtain, and, when disappointed, tramp from one place to another, seeking for work, or return to sit, dejected and sullen, at home. It is not much to be wondered at that they take refuge in the Cup of Lethe, nor that their overtaxed women often do so also. Their bodies relaxed by enforced idleness and undermined by insufficiency of food and by drink, the men contract phthisis in smaller numbers than they did at an earlier period, but still in numbers far too large, and they die at a comparatively early age, leaving their ill-instructed and often feeble wives to rear the children on impossibly small means. If phthisis has invaded the household and spreads to one after another, the curse of poverty and inefficiency is handed on from generation to generation.

With the recurring cycles of trade depression large sections of workmen are thrown out of employment, and become, at least for a time, casuals, undergoing the demoralization of mind and body which results from absence or intermittency of employment. It is true distress is not now so widespread as it was fifty years ago, when all classes suffered in a measure, but the evil of casual labour, the great recruiting officer of the public-house and the common lodging-house, has immensely increased.

It is, above all, intermittency of employment in one shape or another which leads to disease in men, women, and children of the poorest class. It is true this intermittency of employment is deliberately adopted by large numbers of men, vicious, lazy, or defective. But economic conditions are, also, largely responsible, and it is the more normal man who suffers to excess, and whose children suffer.

It is quite evident that poverty leads to disease, partly through malnutrition, partly through overcrowding, partly through neglect of the rising generation, partly through associations and increase in drinking. Drinking, also, in various ways, plays a large part. But alcohol is, I think, not so injurious to the employed man, who works it off, as to the unemployed workman or the drinking woman. Of the results of impoverishment, malnutrition is the most serious, and it is therefore needful to inquire on whom the stress of malnutrition falls most acutely.

There can be no question at all that, under existing social conditions, this stress falls most heavily on widows and on the children of widows. Hence disease is the cause of the most intense poverty. Can it be said conversely that poverty is, of necessity, the most intense cause of disease? It is doubtful. Still, we have seen that amongst the poorest persons there are large numbers who live in common lodging-houses, which are breeding-places of disease. Poor persons suffer from many diseased conditions which they have no occasion to contract.

There are many poor households in which small, too small, wages are earned, but in which good conditions of cleanliness and health are maintained. Let the inmates, however, step outside narrow limitations of habit and associations, and they are at once in peril. On the other hand, below the grade of the skilled artizan, and in the case of many skilled artizans, let the head of the household be cut off, and the family is at once lowered to a degree of poverty which infers malnutrition and consequent degeneration and disease. Disease, therefore, when it affects the head of the house, and often also when other members are affected, implies poverty in a measure and with an amount of precision which does not apply to the influence of poverty in leading to disease. Nevertheless, although this be true, it is also true that poverty, as measured by pauperism, from whatever cause it may arise, is, under existing conditions of life, the cause of much disease both in town and country.

The immediate causes of pauperism may be enumerated as follows:—

(1) Old age. It is evident that this is one of the most fruitful causes of pauperism, especially in rural districts. It affects the large

class who have not been able to save enough to support themselves, and who have no children to take care of them. There is amongst the aged poor, as a class, the greatest reluctance to enter the workhouse, and they have therefore often to submit to overcrowding and to the most undesirable associations. Consequently, they are often exposed to conditions which result in disease. It may be for this reason that the remarkable contrast is exhibited between the proportion of the aged poor and the proportion of other old people who succumb to phthisis.

(2) Illness is perhaps, in large towns, a greater cause of poverty. Fatal illness, by removing the head of the household, frequently leaves the widow with a family for whom she is unable to provide, and who are deeply injured in health and habits by the enforced absence of her care, as well as by physical privation. Or the man loses his work through an attack of pneumonia, pleurisy, enteric fever, kidney disease, or other illness, and, if not quite a first-class workman, is not able to regain his footing. Or, again, he is assailed by a chronic illness which has much the same result as if he succumbed. Perhaps he is the victim of some accident which does not render him penniless, but lowers the earnings of the family below subsistence point; perhaps illness in his family adds to his expenses and prevents any addition to his income. In many ways illness and death in his family impoverish not only the sufferer but his dependents.

(3) Poverty is often the result of incapacity and idleness, both in the married and unmarried. In the unmarried these often lead to association with the common lodging-house and the public-house; in the female too often to prostitution. In a sense the inhabitants of common lodging-houses, as a class, are idle. Their mode of life leads almost inevitably to disease. The married loafer, while himself suffering in a minor degree, causes disease in his wife and children. There is also a considerable number of persons, both married and unmarried, whose work is not worth more than an inadequate wage. Often these are of feeble organization, and so readily fall a prey to illness.

(4) A vast amount of poverty is entailed by the casualization of labour, especially in seaports, but also where there are numerous warehouses or large concerns, such as ironworks, which take on a number of hands by the day. Manchester suffers severely from this source of impoverization. This form of poverty bears with especial severity on the women and children. Associated with this is the employment of boys in wage-earning pursuits which end in the morass of casual labour, the boys finding themselves without employment and without training at

the age of 18 or 19. The stronger marry and add to the ranks of the underfed. The weaker succumb or are drawn into a less reputable mode of life.

(5) Associated with this cause of pauperism in its results is intemperance. There are no doubt many artisans who drink heavily and yet continue to do excellent work, and who keep their places, though their wives and children often suffer. But there are others whose constitutions are wrecked and who become ill, or who become careless and are discharged. These are thrown into the struggling mass of casual labour, from which redemption is nearly hopeless, for a very high proportion of the casual labourers drink. Those in work treat those who are less fortunate, and the man when he is not engaged in looking for work may be found about a public-house, which is his club and centre of information, the place where he is not made uncomfortable, and may receive consideration. It may be stated broadly that intermittent workers drink. Navvies as a body drink. They are often subjected, also, to conditions of crowding. Persons employed in seasonal occupations are apt to drink to excess. There is also a strong tendency in the same direction in workmen exposed to hot employments, as in ironworks and glassworks. The effect of such employments, however, is but partial; of intermittent work it is general. The conditions of ill-health due to impoverishment are thus reinforced by those due to alcohol, and it may be assumed that the effects of drinking are more severe in the under-employed or intermittent worker than in the regular workman. All forms of intermittent and irregular labour are disastrous, and amidst a multitude of improvements they stand out as the impoverishing evil of modern times. Messrs. Rowntree and Sherwell estimate that on an average the weekly expenditure on drink in a working man's family is 6s. This extravagance, which is unfortunately greatest amongst those who can least afford it, paralyzes the progress physically and mentally of a large section of the working class. It is, however, an extravagance which other measures of improvement will gradually abate. This is not, however, the only extravagance which causes impoverishment. There are others—gambling, sexual immorality, vicious and ungenial tempers, which also produce poverty. But they are as nothing in their aggregate magnitude when compared with drinking.

(6) Allied to incapacity, but not identical with it, is ignorance, especially on the part of women. There are many men and women who rise above the defects of their education, and expend their means wisely and well. There is a considerable residuum incapable of instruction,

retention, or initiative whom no help can avail. But there is also a vast amount of discomfort, privation, and disease which might be averted by a rational training of young men and women in the hygiene of ordinary life.

(7) In addition to the causes of impoverishment which have been mentioned, there is one which until recently has been regarded with something of the feeling which attaches to a cyclone or a typhoon. The trade cycle, like the wave of scarlet fever, comes with a fair amount of regularity, but with no diminution of volume, crippling trade unions and friendly societies, and thrusting large masses of labourers into the casual ranks, with all the misery attendant.

(8) More constant in their action is the inheritance of unwholesome dwellings, dark streets, and unbroken areas of dismal homes which we have inherited from an unbridled past, the common lodging-house, and collections of unsavoury refuse. Great, also, have been the evils, both social and sanitary, connected with the circumstances and conditions of individual employment. In no sphere of action, however, has the improvement been greater than in this, though there is still much room for advance.

By the strenuous work of the past generation in the removal of the grosser sanitary evils, and by the long-continued and beneficent work of factory legislation, there has been effected a reduction of mortality which could not fail greatly to reduce pauperism. Still greater has been the result of the enormous progress in medicine and surgery, which has enabled our great public hospitals and our medical practitioners to effect a saving of life and health far beyond the art of half a century ago, and in this way also to reduce poverty. We thus seem to move in a circle. But the reduction of poverty has been due largely to other factors besides those acting through reduction of disease.

The development of machinery brought with it as a direct consequence higher wages, greater freedom of movement from place to place, facility of migration, the products of all countries, cheaper and more varied food for our towns, and, though more slowly, better dwellings and healthier modes of life. In no parts of the country did the wave of prosperity which set in about 1870 fail to be experienced. The congestion and overcrowding of the rural districts was relieved, and the well-being of the labourer both directly and indirectly furthered. Bad conditions were created in many of the large towns, but the relief from impoverishment more than counterbalanced partial retrogressions.

With the general improvement in nutrition the general health was

bound to improve. In the quinquennium 1871-5, it is true, the improvement in the death-rate was not so marked as we should have expected. But in those years prices were high. In 1876 to 1880, when prices had fallen and wages risen, the fall in the death-rate of adults was most striking. It is not, however, till the next quinquennium that we witness a marked fall in the death-rate at ages 0-5, a fall which appears to show that the improvement of the death-rate in the previous quinquennium implied an improved physique.

If we consult the decennial supplements of the Registrar-General we shall find that the improvement in the death-rate in 1871-80 was not confined to urban counties, despite the drain of young people into the large towns. It will be generally conceded that the great advance which then took place in the national health was due mainly neither to advance in medicine nor to fresh administrative achievements, but to material advance. But, if that is so, a direct answer is given to the query whether it can be shown that reduction of poverty reacts at once on the public health.

It is in the experience of every observer of life in our great towns that the poorest classes suffer much more heavily from disease and loss of life than do the better class of artisans or the wealthier classes. It does not follow that their poverty is the cause, although it may be taken for granted that malnutrition produces disease. These classes may be poor through sickness. If, however, we find that the children of the poorest classes suffer in a much higher degree than do those of the better-off classes from all those conditions which are strongly influenced by care, good food, and good clothes, we shall be almost in a position to say that poverty passing a certain limit causes disease. How much may be done by poor people in warding off disease can be seen in the poorest quarters of our large cities, where we can find numerous clean homes and healthy children. But in these poverty is not intermittent, nor does it exceed a certain limit.

From these homes, and from a study of rural life, we are in a position to say that, provided work is steady and certain and wages not too low, poverty in itself need not be a cause of disease. In the aggregate, however, poverty leads to disease in a degree rapidly increasing when we reach the zone marked off by casual labour. Nor, in this regard, does the country differ from the town, though neither poverty nor disease attain in the country that intensity which they assume in large towns.

In arriving at the conclusion that the condition of the children may

be taken as a rough gauge of the effects of poverty, we appear to make the large assumption that children are born equally healthy in the poorest and richest classes. This, of course, is not absolutely true. It is, however, sufficiently near the truth, as is shown by the rapid improvement in physique which infants show when taken to a well-ordered hospital or crèche, and by the great improvement of the physique of poor children when suitably provided for under the Poor Law.

For a determination of the degrees of poverty prevailing in a community, and of the relative influence exerted by different causes of impoverization, there is ultimately no other way than to make an investigation into the conditions of every household. We are all familiar with the very complete inquiry instituted by Mr. Seeböhm Rowntree, following in the footsteps of Mr. Charles Booth, into the circumstances of the working-class population of the City of York, made at a period when work was more than usually abundant. Mr. Rowntree divided the working-class population into four sections: (A) Total family income under 18s. for a moderate family; (B) Total family income 18s. and under 21s. for a moderate family; (C) Total family income 21s. and under 30s. for a moderate family; (D) Total family income over 30s. for a moderate family. By a moderate family is intended a family consisting of father, mother, and from two to four children, allowance being made for the size of the family in classifying each. The following are particulars ascertained with regard to each of the classes in question:—

	Number of persons in each class	Percentage of working-class population	Percentage of total population	Average size of family	Average family earnings of families earning anything	Average family earnings
					s. d.	s. d.
A	1,957	4·2	2·6	3·00	11 7	8 4½
B	4,492	9·6	5·9	4·56	—	19 9
C	15,710	33·6	20·7	4·11	—	26 7
D	24,595	52·6	32·4	4·03	—	41 9½

He then divides the absolute necessities of life into food, rent, and household sundries. The household sundries are carefully estimated, and the food is calculated on a scale put forward by Atwater, which cannot be regarded as over-generous. He then adds up the total income and total expenditure of each family, and on a comparison arrives at the conclusion that 9·91 per cent. of the total population of York are in a state of primary poverty; that is to say, have not means enough to defray the necessary elementary expenses. He further frames an estimate from the

observed condition of the families of the total number of persons living in a state of poverty, which he places at 27·84 per cent. of the total population. By deducting the 9·91 per cent. living in primary poverty he obtains a figure for what he calls secondary (i.e., unnecessary) poverty—viz., 17·93 per cent. of the population.

What we are here concerned with is the immediate cause which he gives for impoverishment. The causes which he assigns to Class A, which answers to our destitute class, make it appear that in the City of York in the case of 1,295 persons, or almost exactly two-thirds of the whole, the immediate cause of poverty is removal of the wage-earner by death or desertion, or the inability to earn wages through illness or old age. "Economic" causes account for 418 persons, or about 21 per cent. In the direct production of this degree of destitution, then, disease plays the largest part.

So far as Manchester is concerned, these figures undoubtedly greatly understate the immediate influence of casual labour and of want of work, even at the best of times, in producing destitution; but it is to be remembered that the period during which the inquiry was made by Mr. Rowntree was one of exceptional prosperity. It is, however, most valuable evidence of the results of disease in such communities as York, and in a period of abounding prosperity.

The conditions of three small districts in Manchester of the poorest character were inquired into for the Committee on Physical Deterioration. In one of these the causes of impoverishment were much the same as Mr. Rowntree found in York. In the other two the predominant factor was small wages; that is to say, casual labour and incapacity. Nevertheless, in all three disease played a prominent part. It is impossible to disentangle disease from economic factors, since, of course, the primary factor concerned with the death of the husband may have been casual labour.

Mr. Rowntree does not classify the causes of impoverishment in the Classes B, C, and D separately. He does, however, classify the causes for the 9·91 per cent. of the population suffering from primary poverty. Sickness now takes a secondary, though still an important, place. The conditions of labour preponderate. It is possible that of the 51·96 per cent. impoverished by low wages, a certain proportion, though in regular work, do not work full time. Otherwise we have here a very large factor for which no immediate remedy is possible. This applies, also, in large measure to the 22·16 per cent. impoverished by the size of the family. It may be assumed that this is pre-eminently the class who will not apply for assistance.

In this population, then, there was apparently a total of 74 per cent. of the impoverished class not readily accessible to any system of public aid, but who, it may be assumed, suffer heavily from disease. The table shows very clearly why poverty has so much declined, since (with the exception of casuals and, perhaps, of persons out of work) it is certain that each of the classes here dealt with has greatly declined relatively to the whole population.

Taking three divisions of the working-classes, which he calls poorest, middle, and highest, Mr. Rowntree obtains for the year 1898 the following figures:—

Area	No.	1	...	Death-rate	...	Birth-rate
		1	...	27·78	...	39·83
		2	...	20·71	...	40·32
		3	...	13·49	...	29·00

These figures serve to show that the most severe poverty here dealt with was far above the average of poverty in the Manchester Township. They may suffice, however, to suggest the part which even a moderate excess of impoverishment takes in the production of a high death-rate.

Mr. Charles Booth, in his great work, "Life and Labour of the People," published in 1892, classified the poorest sections of the working classes as follows:—

			Percentage of total	
(A) The loafers and wastrels and their families	1·23	} Very poor
(B) Casual earnings	11·22	
(C) Irregular earnings	8·33	} Poor
(D) Regular minimum	14·46	

He gives the following analysis of the immediate causes of poverty:—

Great poverty (Classes A and B)		Percentage of total	Poverty (Classes C and D)		Percentage of total
1	Loafers	4	1	Loafers	—
2	Casual work	43	2	Low pay (regular earnings)	20
3	Irregular work, low pay	9	3	Irregular earnings	43
4	Small profits	3	4	Small profits	5
5	Drink (husband, or both husband and wife)	9	5	Drink (husband, or both husband and wife)	7
6	Drunken wife	5	6	Drunken wife	6
7	Illness or infirmity	10	7	Illness or infirmity	5
8	Large family	8	8	Large family	9
9	Illness or large family, combined with irregular work	27	9	Illness or large family, combined with irregular work	19
		9			5
Total	...	100	Total	...	100

It will be seen that over the great area of North-East London which he investigated, with a population of about 900,000 persons, he found the influence of occupation dominant. Illness is here also an important factor. It is probable, in his view, that incapacity figures very largely in the production of the casual and irregular classes, as shown by lack of ability to produce a high standard or amount of work, or to remain at work.

We may assume, then, that pauperism measures a degree of destitution below want, and that those actually in want of the barest necessities of life in the poorest parts of our large towns cannot be placed below 10 per cent. Their impoverishment is in the main due to immediate loss or deficiency of work and to illness. Behind both causes lies the determining factor of relative incapacity in one or another form which affects a great part of those who become destitute. This relative incapacity extends to their physical condition and in part determines an excess of disease among the very poor. The greater part of the excess of fatal illness from which they suffer, however, appears to be determined otherwise, and to be conditioned by malnutrition, personal habits, relatively insanitary housing, overcrowding, and ignorance.

The problems of poverty and of the disease consequent upon it oblige us to divide the poor into two classes: those who are poor through misfortune or fault but who are capable, and those who fall into poverty through incapacity. For the first class we need education and organization; the second presents a much more complex problem.

I propose now to consider the subject in the light of the experience of Manchester.

PART II.

THE RELATION OF POVERTY TO DISEASE IN MANCHESTER.

What light can be shed on the relation of poverty to disease by a study of the vital statistics of Manchester? I have found it impossible within a brief space or without tables to convey the picture which one has so far been able to form. Yet a complete statement and the presentation of these tables would have extended this address beyond all reasonable length. I have been obliged, therefore, simply to indicate without enlarging on the arguments which lead to the conclusions arrived at. These conclusions, or perhaps rather convictions, may be thus stated: As poverty deepens, the death-rate rises at every age group;

this difference in the death-rate is due mainly to impoverishment and its attendant evils. The diseases which respond most clearly to differences of social position are in childhood those in which the fatality depends most intimately on good nourishment and instructed care, and particularly diseases of the digestive and respiratory systems; in later years it is lung disease which responds clearly and unmistakably to poverty. It is, however, tubercular phthisis which shows the sharpest reaction and the widest differences. The important factors in the production of the wide differences observed in males appear to be life in common lodging-houses and the extent of casual labour. Phthisis is in the main generated in Manchester, and the disease in common lodging-houses is for the most part generated in them.

Nearly one-half of the male mortality of Manchester from phthisis occurs in public institutions, and the influence of segregation is exerted on the largest scale. It is, however, unable to do more than cope with the incoming stream of phthisis added to the numbers produced in the central parts of the city. The problem is largely a social one; but the stringent regulation and supervision of common lodging-houses is also indicated.

Manchester is divided into three parts—a central part, the Manchester Township, South Manchester, and North Manchester. South Manchester contains far the largest part of the Chorlton Union, North Manchester far the largest part of the Prestwich Union. The figures of pauperism for the three unions sufficiently indicate the extent of destitution in each division, and show that there is much more destitution in the Manchester Township than in South Manchester, and considerably more in South than in North Manchester.

The proportion of deaths occurring in the union hospitals is another measure of destitution which corroborates the figures for pauperism.

The Manchester Township is divided into three sanitary districts—Central, St. George's, and Ancoats. The Central has most common lodgers in proportion to population, and has considerably the highest death-rate from phthisis; St. George's comes next in both respects, and Ancoats last.

South Manchester is divided into eight sanitary districts, of which two were included in 1904 and do not appear in this analysis. Of the older sanitary districts, Hulme and St. George's have a considerable area of very poor persons in the portion nearest to the Town Hall.

North Manchester, with the exception of a small portion of Cheetham, nearest to the Town Hall, has no very poor areas. North Manchester is

divided into nine sanitary districts, the populations of which are mostly smaller than those of the other districts.

Figures are here inserted in illustration of the above observations.

MANCHESTER, 1901-03.

Statistical divisions and districts	Census populations, 1901	Death-rates of persons dying at home	Death-rates of persons dying in union hospitals	Death-rates of persons dying in institutions	Total death-rates	Percentage of deaths occurring in all institutions
Manchester City ...	546,408	15·46	3·06	1·84	20·36	24
(1) Manchester Township	135,006	16·98	6·26	2·74	25·98	35
(2) North Manchester ...	167,257	14·23	0·91	1·53	16·67	15
(3) South Manchester ...	244,145	15·48	2·80	1·58	19·86	22
Manchester Township:						
Ancots ...	45,014	18·44	4·78	3·18	26·40	30
Central ...	30,047	15·30	9·28	3·10	27·68	45
St. George's... ..	59,945	16·71	5·88	2·22	24·81	33
North Manchester:						
Cheetham	37,207	10·70	0·76	1·67	13·13	19
Crumpsall	8,852	11·63	0·86	1·32	13·81	16
Blackley	8,873	13·70	0·74	1·59	16·03	15
Harpurhey	15,893	13·89	0·66	1·11	15·66	11
Moston	12,161	12·19	0·20	1·07	13·46	9
Newton	40,525	15·49	1·11	1·49	18·09	14
Bradford	23,766	18·41	1·42	1·62	21·45	14
Beswick	11,686	16·86	1·15	2·17	20·18	16
Clayton	8,289	12·89	0·66	1·61	15·16	15
South Manchester:						
Ardwick	41,454	15·72	1·98	1·81	19·51	19
Openshaw	27,358	16·10	1·52	1·36	18·98	15
West Gorton	29,459	14·22	1·97	1·51	17·70	20
Rusholme and Kirkmanshulme	20,544	14·23	1·45	1·17	16·85	16
Chorlton-on-Medlock	*57,956	13·75	3·93	1·54	19·22	28
Hulme	67,374	17·61	3·73	1·73	23·07	24

The Manchester Township is flat, and lies on the chief streams. With the exception of its hotel population, it is generally inhabited by the poorest class of labourer, largely casuals, and by persons engaged in the lowest grades of work, with those who provide for their requirements.

South Manchester has a considerable element of the same kind. Further out are clerks and persons of that rank. There is also a considerable residential and business population. The district is flat.

North Manchester is inhabited principally by artisans, in portions by common labourers. There is also a fair number of residential houses in the outer portions of Cheetham and Crumpsall. This portion of the

City is at a higher level than the two other divisions; the surface is more undulating, and the drainage of the soil more efficient.

Manchester is very rich in voluntary hospitals and charitable institutions. There are also a number of shelters and homes for working men. Unquestionably these exert a strong attraction on men down on their luck and, though to a less extent, on the sick. Institution deaths constitute, at present, 26 per cent. of the total.

In the Manchester Township institutional treatment is liberally accorded and out-relief is discouraged. In South Manchester out-relief is more freely given, as is also the case in North Manchester. Up to recently hospital provision in North Manchester under the Poor Law was inadequate.

The percentages of deaths in each division occurring in public hospitals—voluntary, union, and municipal—were for the years 1901-3 : Manchester Township, 35 ; South Manchester, 22 ; North Manchester, 15.

The following statements made with regard to the classes of disease which respond most readily to poverty are founded on an examination of two sets of statistics for the periods 1891-3 and 1902-4 respectively. Each gives the death-rates for three years at each of the six groups from a number of causes of death, for each of the three main divisions of the city. The figures are in entire accord in marking out diseases of nutrition in childhood, and at every age-group diseases of the lung, as reflecting the influence of poverty. The reaction of phthisis to social conditions is even startling. At ages under 5, measles, whooping-cough, and diarrhoea also reflect the influence of poverty, the last-named very markedly.

Having regard to the influence which must be exerted by our great hospitals in reducing mortality and disablement, as well as in the removal of infection, I have analyzed for one year—viz., for 1903—the fatal disease treated in the union and other hospitals. The results may be thus stated : At ages below 5, 10 per cent. of all deaths occur in public hospitals ; at ages above 5, more than one-third of the deaths occur in these institutions ; above the age of 65, however, the proportion is somewhat less, though there is no great difference in the percentage at different age periods above the age of 5 years. The proportion occurring in the union hospitals increases after the age period 5—14 up to advanced ages, at first rapidly ; after the age period, 25—44, slowly. The highest proportion in other hospitals is at the age period 5—14, diminishing gradually as age advances.

As regards the proportions of deaths from individual causes occurring in public hospitals, considering only those which yield a large number of deaths, the highest occur under the heads of diseases of the nervous system, with a percentage of 62·8 among males and 51·4 among females; phthisis, with 45·6 per cent. of males, 33·9 of females; and a group including diseases of the circulatory system, bronchitis, pneumonia, diseases of the digestive system, and diseases of the urinary system, which yield, severally, about one-third of the male deaths, but a considerably lower proportion of females.

Deaths from malignant disease give 49·7 per cent. males, 24·2 per cent. of females, and violence yields 56·1 per cent. males and 56·6 per cent. females. The two last-named causes may be excluded from consideration. In union hospitals the highest percentage is under phthisis—41·9 per cent. males, 30·5 per cent. females. Diseases of the nervous system include 35·5 per cent. males, and 27·4 per cent. females. Bronchitis gives 33·3 per cent. males, 10·9 per cent. females. Diseases of the heart and blood-vessels furnish 26·4 per cent. males, 17·7 per cent. females. From pneumonia we get 26·6 per cent. males, 27·5 per cent. females. The rest may be disregarded, giving, as they do, much lower percentages and a much smaller number of deaths.

Diseases of the nervous system cannot be said to show the influence of poverty so much as to be a cause of poverty. To some extent also this is true of phthisis—say to the extent of 20 to 30 per cent. of the cases. Of the remainder, also, it is often true that what causes their poverty causes phthisis, inasmuch as instability on the part of the brain-cells doubtless goes along with instability of the tissue-cells. Demonstrably, however, under certain conditions produced by poverty, the strongest frames yield to the infection. In regard to bronchitis, and still more in the case of heart diseases and pneumonia, alcoholism steps forward as a cause in association with poverty, which it produces or aggravates. The excessively high death-rate from pneumonia in Manchester may point to excessive intemperance, and it is the case that Manchester has the largest proportion of public-houses to population of all large centres. Whether, however, excessive intemperance is due to poverty, or poverty is more due to intemperance, is a difficult question. We have no means of properly gauging the amount of intemperance.

Clearly phthisis is the most convenient mirror of poverty. In its production such factors as malnutrition, alcoholism, overcrowding and uncleanness take an important share, and these are in special

association with poverty. In order to study more nearly the relation of phthisis to poverty, I have calculated out for the years 1901-5, in six groups of ages, for both sexes, the death-rates from phthisis occurring in each sanitary district among persons at their own homes, in the union hospitals, and in other institutions. Before giving this table, however, it is needful to ask ourselves what the tables can be taken to represent? Are the deaths dealt with those of persons who have contracted their disease in the districts, or have the persons dying in a district, or in institutions, but referable to the district, contracted their disease elsewhere? The answer to this question requires a laborious examination of the investigation sheets of notified cases, a procedure which involves the expenditure of much time and care. So far it has been possible to carry out such an inquiry in respect only of one year—viz., 1907. Mr. G. H. Lock, Chief Clerk of the Tuberculosis Department, has exhibited for this year in three tables, one for each of the main divisions of the city, the sanitary district from which each case has been notified, whether directly or from the union hospitals, with the district in which the patients were living when infected. The results may be summarized thus:—

TOTAL CASES FOR THE MANCHESTER TOWNSHIP, 1907.

Cases infected while living in the Division	419
" " " North Manchester	28
" " " South Manchester	25
" " " outside Manchester	114
Tramps	49
Total	635

TOTAL CASES FOR NORTH MANCHESTER, 1907.

Cases infected while living in the Division	165
" " " the Manchester Township	23
" " " South Manchester	12
" " " outside Manchester	48
Total	248

TOTAL CASES FOR SOUTH MANCHESTER, 1907.

Cases infected while living in the Division	306
" " " the Manchester Township	16
" " " North Manchester	15
" " " outside Manchester	93
Total	430

Thus, for this year, so far as we could ascertain, about two-thirds of the cases notified from the central and poorest division were produced while they lived in the centre; rather more than two-thirds of the South

Manchester cases were produced during residence in that division; and in North Manchester also about two-thirds had their origin during residence in the division.

There is, therefore, no such drift of poor phthisical persons from the outer parts of Manchester into the centre as we might expect. On the other hand, 255 persons notified out of the total 1,315 here considered contracted their disease outside, in addition to 49 tramps. This gives rather less than one quarter of the total notified phthisis as imported. So far as we can judge, there is no corresponding amount of exported phthisis; but this cannot be determined easily, if at all. Cases of phthisis, therefore, move into the union hospitals of their own divisions, and do not drift into the central and poorer part of the city. This is similar to the conclusion at which I arrived during the investigation of deaths in Oldham twenty years ago. These figures, however, make it necessary to allow considerable deductions from any conclusions arrived at from a study of the statistics of mortality for individual districts, especially in the smaller districts of North Manchester.

Mr. Lock has also handed me a summary of facts concerning 6,942 cases notified in the five years 1903-7 which he has drawn up in elucidation of the question of drift. He finds that of these cases 2,200 were admitted into the union hospitals from private addresses and 1,170 from common lodging-houses. The number of cases reduced during that period from private addresses to common lodging-houses, presumably as the result of disease, and which, having been admitted into union hospitals, were notified to the Public Health Office, was 224, or 3·27 per cent. of the total cases notified.

The amount of drift of phthisical persons into common lodging-houses is not large, and emphasises the view long held, that the phthisis of common lodging-houses is produced in this class of habitation, and may be considered apart from that of the poorer population generally. This view is entirely borne out by our individual histories of cases of phthisis notified from union hospitals and admitted into these from common lodging-houses. Practically the whole of the common lodging-house cases come to us through notifications from the union hospitals. Many of them have lived for many years in these places, though, after contracting phthisis, they usually go downhill rapidly.

Before going on to consider more closely the relation of poverty to phthisis, we may now direct our attention to the detailed table of death-rates, constructed in the manner which I have described (*see pp. 22-25*).

We note first the very large difference between the male and female death-rates, amounting to 1·9 per 1,000 in the Manchester division, to 1 per 1,000 in South Manchester, and only to 0·26 in North Manchester.

DEATH-RATES FROM

Statistical Divisions	DEATHS OCCURRING AT HOME							DEATHS OCCUR-	
	Age 0-4	Age 5-14	Age 15-24	Age 25-44	Age 45-64	Age 65 and over	All ages	Age 0-4	Age 5-14
City of Manchester ...	0·296	0·218	1·079	2·085	2·849	1·090	1·350	0·018	0·049
(1) Manchester Township ...	0·285	0·345	0·947	2·391	3·067	0·754	1·489	0·026	0·105
(2) North Manchester ...	0·262	0·149	1·045	1·712	2·086	0·678	1·062	—	0·021
(3) South Manchester ...	0·327	0·203	1·172	2·182	3·193	1·575	1·475	0·028	0·041
Manchester Township :									
Ancoats ...	0·353	0·512	0·970	2·230	3·132	—	1·436	—	0·043
Central ...	—	0·386	1·154	2·159	3·205	2·381	1·595	—	0·154
St. George's ...	0·333	0·194	0·803	2·579	3·231	0·948	1·495	0·055	0·129
North Manchester :									
Cheetham ...	0·090	0·050	1·079	1·128	1·684	2·126	0·848	—	—
Crumpsall ...	—	—	0·994	0·900	0·342	—	0·529	—	—
Blackley ...	—	0·217	1·235	3·024	2·062	—	1·509	—	—
Harpurhey ...	0·336	—	1·184	2·038	2·618	—	1·231	—	0·108
Moston ...	—	0·267	0·713	1·975	2·296	—	1·097	—	—
Newton ...	0·354	0·097	0·905	2·156	2·947	1·039	1·291	—	0·049
Bradford ...	0·118	0·421	1·097	1·880	2·656	—	1·181	—	—
Beswick ...	1·152	0·148	1·812	1·068	0·876	—	0·965	—	—
Clayton ...	0·282	0·167	0·665	0·977	0·758	—	0·577	—	—
South Manchester :									
Ardwick ...	0·390	0·174	1·031	1·932	2·582	2·367	1·259	—	0·044
Openshaw ...	—	0·066	1·195	1·706	1·782	0·627	1·005	—	0·066
West Gorton ...	0·297	0·058	1·272	2·239	3·452	—	1·381	—	—
Rusholme and Kirkmans-	—	0·097	1·128	1·776	3·137	2·247	1·331	0·147	—
hulme ...	0·390	0·123	1·178	2·296	2·938	1·646	1·549	0·078	0·082
Chorlton-on-Medlock ...	0·510	0·441	1·233	2·671	4·408	2·332	1·861	—	0·029
Hulme ...	—	—	—	—	—	—	—	—	—
Moss Side ...	—	—	—	—	—	—	—	—	—
Withington ...	—	—	—	—	—	—	—	—	—

There must clearly be some peculiar circumstances connected with these discrepancies. Clearly it cannot be housing as far as ordinary houses or tenements occupied by families are concerned. Taking Manchester as a whole, the female phthisis death-rate must reflect the conditions of housing, while at the same time reflecting the infection due to the high

phthisis rate in men. We may conclude that the relatively high incidence of phthisis in males in Manchester is, for the most part, not due to housing conditions.

PHTHISIS, MALES, 1901-05.

RING AT THE UNION HOSPITALS					DEATHS OCCURRING AT THE OTHER HOSPITALS							Grand Total, all ages
Age 15-24	Age 25-44	Age 45-64	Age 65 and over	All ages	Age 0-4	Age 5-14	Age 15-24	Age 25-44	Age 45-64	Age 65 and over	All ages	
0.272	1.425	3.133	2.243	0.982	0.062	0.056	0.081	0.182	0.120	—	0.107	2.439
0.637	3.493	7.668	6.889	2.549	0.104	0.075	0.093	0.264	0.164	—	0.150	4.188
0.058	0.217	0.331	0.113	0.124	0.037	0.064	0.081	0.145	0.136	—	0.096	1.282
0.229	1.137	2.193	0.501	0.727	0.057	0.041	0.074	0.162	0.084	—	0.092	2.294
0.508	2.652	5.731	5.366	1.788	—	0.128	0.092	0.442	0.200	—	0.204	3.428
0.747	4.361	11.586	12.500	3.661	—	0.077	0.065	0.220	0.247	—	0.140	5.396
0.664	3.610	7.471	10.111	2.515	0.222	0.032	0.105	0.198	0.101	—	0.117	4.127
0.047	0.255	0.532	—	0.150	0.090	0.050	0.235	0.120	0.177	—	0.161	1.159
—	0.150	—	—	0.048	—	—	—	—	—	—	—	0.577
—	0.288	—	—	0.091	—	—	—	0.144	0.344	—	0.091	1.691
0.132	0.146	0.201	—	0.118	—	0.108	—	0.073	—	—	0.047	1.396
—	—	—	—	—	—	—	—	0.180	—	—	0.058	1.155
0.106	0.260	0.253	—	0.142	0.089	0.097	0.053	0.074	0.168	—	0.088	1.521
0.078	0.243	0.978	—	0.200	—	0.140	0.078	0.243	0.140	—	0.133	1.514
—	0.475	—	1.835	0.172	—	—	—	0.119	—	—	0.034	1.171
—	—	—	—	—	—	—	—	0.140	0.379	—	0.082	0.659
0.328	0.717	1.595	—	0.509	—	—	—	0.218	0.304	—	0.106	1.874
—	0.487	1.114	—	0.306	—	0.066	0.075	0.098	—	—	0.058	1.369
0.201	0.685	1.448	—	0.416	0.099	0.058	0.067	0.365	0.111	—	0.161	1.958
0.087	0.209	1.004	—	0.235	—	—	0.260	0.157	—	—	0.105	1.671
0.277	1.811	3.014	—	1.153	—	0.041	0.104	0.095	0.096	—	0.082	2.784
0.285	1.670	3.110	1.166	1.018	1.153	0.059	0.032	0.104	—	—	0.070	2.949
—	—	—	—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	—	—	—	—	—

The differences between the male and female death-rates are greatest in the three districts of the Manchester Township and in Chorlton-on-Medlock. These are the districts of common lodging-houses. Is it possible that common lodging-houses should make so great a difference? In 1899 the Chief Constable made an enumeration of the persons living

in common lodging-houses, and found these, male and female, to number 5,831. Since then a number of the smaller places have been closed, but a number of other places have been put on the Police Register,

DEATH-RATES FROM

Statistical Divisions	DEATHS OCCURRING AT HOME							DEATHS OCCUR-	
	Age 0-4	Age 5-14	Age 15-24	Age 25-44	Age 45-64	Age 65 and over	All ages	Age 0-4	Age 5-14
City of Manchester ...	0·214	0·322	1·014	1·520	1·203	0·317	0·941	0·024	0·081
(1) Manchester Township ...	0·280	0·398	1·046	1·814	1·574	0·177	1·096	0·025	0·162
(2) North Manchester ...	0·150	0·334	1·036	1·422	1·097	0·225	0·882	—	—
(3) South Manchester ...	0·224	0·273	0·981	1·445	1·073	0·440	0·901	0·042	0·096
Manchester Township :									
Ancoats ...	0·321	0·251	1·137	1·872	1·475	0·607	1·076	—	0·084
Central ...	0·270	0·607	0·855	1·158	1·306	—	0·884	—	0·228
St. George's ...	0·326	0·404	1·042	2·062	1·828	—	1·199	—	0·187
North Manchester :									
Cheetham ...	0·171	0·052	0·616	1·251	1·097	—	0·685	—	—
Crumpsall ...	—	—	0·753	0·754	—	2·062	0·694	—	—
Blackley ...	—	0·799	0·859	2·575	2·026	—	1·417	—	—
Harpurhey ...	0·358	0·510	1·288	1·135	1·583	—	0·962	—	—
Moston ...	—	0·127	0·934	1·301	0·704	—	0·711	—	—
Newton ...	0·088	0·098	1·141	1·530	0·913	—	0·839	—	—
Bradford ...	0·251	0·626	1·298	1·544	0·637	—	0·959	—	—
Beswick ...	—	1·170	1·660	1·617	1·062	—	1·197	—	—
Clayton ...	0·276	0·180	1·427	1·504	1·463	1·887	1·012	—	—
South Manchester :									
Ardwick ...	0·226	0·452	0·812	1·471	1·478	—	0·936	—	—
Openshaw ...	0·220	0·196	0·728	1·532	0·632	0·452	0·763	—	—
West Gorton ...	0·099	—	1·077	1·163	0·886	—	0·676	—	0·057
Rusholme and Kirkmans-	—	—	1·174	0·772	1·000	0·402	0·681	—	0·090
hulme ...	—	—	—	—	—	—	—	—	—
Chorlton-on-Medlock ...	0·308	0·359	0·820	1·298	0·797	0·690	0·848	0·077	0·079
Hulme ...	0·491	0·432	1·132	2·495	3·685	1·399	1·712	0·098	0·231
Moss Side ...	—	—	—	—	—	—	—	—	—
Withington ...	—	—	—	—	—	—	—	—	—

including the Municipal Home for men, the Wesleyan Home, the Salvation Army Shelters, the Church Army Shelters, the tramp wards. It is now reckoned that there are 7,392 beds for men, 357 for women, and 239 for married couples. Total beds, 7,988 ; total persons provided for, 8,227.

There is also accommodation in houses registered as let in lodgings or occupied by members of more than one family for 16,424 persons. There are no means, however, of determining how these lodgings are

PHTHISIS, FEMALES, 1901-05.

RING AT THE UNION HOSPITALS					DEATHS OCCURRING AT THE OTHER HOSPITALS							Grand Total, all ages
Age 15-24	Age 25-44	Age 45-64	Age 65 and over	All ages	Age 0-4	Age 5-14	Age 15-24	Age 25-44	Age 45-64	Age 65 and over	All ages	
0.256	0.715	0.968	0.824	0.453	0.073	0.049	0.094	0.088	0.034	0.021	0.070	1.464
0.473	1.762	2.609	2.483	1.117	0.127	0.074	0.115	0.114	0.020	—	0.090	2.303
0.095	0.113	0.137	—	0.072	—	0.032	0.127	0.083	0.051	0.075	0.068	1.022
0.255	0.609	0.615	0.484	0.372	0.098	0.048	0.060	0.079	0.031	—	0.061	1.334
0.295	1.185	2.065	3.035	0.804	0.107	0.042	0.126	0.218	0.059	—	0.114	1.994
0.428	2.501	3.638	5.025	1.600	0.405	0.076	0.244	0.093	—	—	0.140	2.624
0.619	1.787	2.605	1.900	1.112	0.054	0.093	0.033	0.046	—	—	0.046	2.357
0.041	0.243	0.439	—	0.139	—	—	0.082	0.104	0.145	—	0.070	0.894
—	—	—	—	—	—	—	—	—	—	1.031	0.041	0.735
0.430	0.136	—	—	0.125	—	—	0.644	0.136	—	—	0.167	1.709
0.107	0.134	0.352	—	0.107	—	—	—	0.067	—	—	0.021	1.151
—	—	—	—	—	—	—	0.267	0.087	—	—	0.082	0.793
0.156	0.107	0.152	—	0.084	—	—	0.104	0.142	—	—	0.063	0.986
0.076	0.119	0.127	—	0.065	—	—	0.153	—	0.255	—	0.065	1.279
—	0.323	0.266	—	0.129	—	0.292	—	0.108	0.266	—	0.129	1.455
—	0.137	—	—	0.040	—	—	0.408	0.273	—	—	0.162	1.214
0.090	0.360	0.269	0.318	0.174	—	—	0.045	0.120	—	—	0.046	1.156
0.073	0.335	0.316	—	0.156	—	0.065	0.291	0.239	0.021	—	0.170	1.089
0.336	0.313	1.083	—	0.312	0.099	0.172	—	0.089	—	—	0.078	1.066
—	0.163	0.100	—	0.087	0.157	0.090	0.069	—	—	—	0.043	0.811
0.498	0.958	0.997	0.517	0.561	0.231	0.040	—	0.060	—	—	0.039	1.448
0.261	0.877	0.970	1.224	0.557	0.098	0.029	—	0.058	—	—	0.035	2.384
—	—	—	—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	—	—	—	—	—

distributed, or in which of them aggregations of men or women occur of a character akin to that which prevails in common lodging-houses. It may be said, generally, that this kind of crowding probably occurs to the greatest extent in the following districts, taking them in their order: Central, St. George's, Hulme, Ancoats, Chorlton-on-Medlock, and Ardwick.

It is better, then, to confine our attention at present to common lodging-houses and to men. The details of distribution of the common lodging-house provision existing in 1908 are as follows:—

ABSTRACTED FROM THE COMMON LODGING-HOUSE REGISTER.

		Number of houses		Number of beds
Manchester Township:				
Ancoats, males	...	18	...	1,238
Central, males	...	55	...	2,129
„ females	...	8	...	194
„ couples	...	2	...	61
St. George's, males	...	24	...	2,142
„ females	...	5	...	90
„ couples	...	10	...	178
				6,032
Number of persons, 6,271				
North Manchester:				
Cheetham, males	...	2	...	198
				198
South Manchester:				
Ardwick, males	...	4	...	148
Chorlton-on-Medlock, males	...	16	...	1,162
„ females	...	2	...	73
Hulme, males	...	8	...	385
				1,768
Total beds				7,988
Total number of persons, 8,227				

On referring to the tables of death-rates from phthisis it will be seen that the death-rates in the union hospitals rise with the amount of common lodging-house provision, small as this is by comparison with the total population. We are in a position to form an estimate of the death-rate in common lodging-houses. From the table supplied by Mr. Lock we find that in the years 1903-7 the number of cases notified as having come from common lodging-houses was one-sixth of the total. Now, the phthisis mortality in the Central division is nearly four-tenths of that in the whole city. Hence the death-rate arising from the common lodging-houses in the township might be reckoned for the Central division at $\frac{1}{6} \times \frac{1}{4}$ of the total, if all common lodging-house beds for males were in the centre. The number, however, is 5,509 out of 7,392. The male death-rate in the Manchester Township may therefore be reckoned as $\frac{1}{6} \times \frac{1}{4} \times \frac{5.5}{7.4} \times 4.188$ per 1,000, or 1.3 per 1,000. This gives a male death-rate in the common lodging-houses, if all the beds were occupied, of $1.3 \times \frac{6,528.8}{5,509}$, or 15.4 per 1,000.

It seemed important, however, to check this estimate. I have therefore had a statement made out for the years 1901 and 1908 of

the deaths in workhouses coming respectively from common lodging-houses and from private houses for each sanitary district in the city. Without entering into further details, I will only say that direct calculation gives for 1908 a death-rate of 15·1 per 1,000 in common lodging-houses, if beds be reckoned as above; if estimated average population be taken, of about 20 per 1,000.

For 1901 I have added 10 per cent. to the common lodging-house population as enumerated in 1899, so as to be certain that the population was not under-estimated. Nevertheless, the death-rate comes out at 20·5 per 1,000 of the common lodging-house population. The common lodging-houses, then, are a focus of infection, which spreads, as I have elsewhere shown, by public-houses, and doubtless also through the markets, warehouses, and other places in which the inhabitants are occupied among the rest of the population. The size of the lodging-house population is no criterion of the amount of phthisis generated in these places.

The same influence is seen in the female death-rates in each district of the Manchester Township, in Hulme and in Chorlton-on-Medlock, though the effects are necessarily much smaller. The impress of the common lodging-houses is to be seen in the death-rates allocated to the union hospitals. Its most marked feature is its tendency to increase up to the highest ages, even above the age of 65. The fierce flame of lodging-house infection burns up the most resistant material. If, however, we take away one half of the male union death-rate as due to life in the common lodging-house in the Manchester Township, we still leave a high union death-rate, as we see from the figures both for males and females, though we have reduced the difference between the sexes to more normal dimensions. Thirty per cent. of the male deaths from the Manchester Township still occur in union hospitals, when the common lodging-house element is removed.

The mode of occurrence of this death-rate can be seen in the figures for Hulme, in which district impoverishment is great, although the common lodging-house element is comparatively small; or in the figures for South Manchester generally. Here we can study the destitution element in the workhouse death-rates, and also the mode of distribution with a less severe degree of destitution, in the home figures of the Manchester Township or of South Manchester. The general feature of the death-rate for Hulme is the steady ascent of mortality up to the age period 45—64 in which it culminates, both in males and females and both inside and outside the union hospitals.

For further elucidation of these figures we may refer to the Registrar-General's figures in the last decennial supplement (p. cc). The maximum incidence of rate of mortality for 1891-1900, we find, was for males in urban counties at the age period 45—54, in rural counties at the age period 25—34; for females the maximum mortality was in urban counties at ages 35—44, for rural counties at 25—34, as in males, but with a greater leaning towards the lower age of 15—24 than in males. Turning now to page cxiii, we find that the death-rate from phthisis fell immensely through four successive decennia, but the age of maximum incidence, instead of receding, has advanced. We may, I think, venture to gloss these figures thus. At former periods England and Wales were more prevailingly agricultural, as in Ireland to-day. Instead of the lakes of stagnant labour which we find in our great towns, there were then numerous ponds and pools scattered all over the country. There was much greater poverty in the country than at present, and much more phthisis in consequence, the least resistant being weeded out at a comparatively early age. As years advanced, however, the healthy country life told on the constitution of the more resistant and enabled them to repel or throw off the disease. So, but in a much higher degree, it is with the agricultural population of England to-day. Not only are there more recoveries, but fewer are attacked, in consequence of the improved conditions.

There is no comparison between the poverty in the country and that in the centre of our great towns under which casual labourers and widows suffer. Here we have the maximum of infection with the minimum of resistance. Broadly speaking, as England and Wales were in 1861-70, Ireland is to-day. Infection occurs at numerous points, there is a general poor level of nutrition and mode of living, but the effect of the healthy country life asserts itself in later years.

This, however, is not the whole of the explanation. There is much more aggregation of human beings in towns than in the country, and hence, under equal conditions of nutrition, there is more infection. Men are aggregated in workshops, and also in public-houses and clubs, at an age when women have ceased to meet. The labour of women is restricted to a comparatively early age. Hence, largely, the higher age of male phthisis-incidence in towns. It is, in the main, a function of work, in so far as work produces aggregation and intercourse.

There is no such separation of the ages of maximum incidence in the country, because there are no such great aggregations of men and women. The relatively high incidence of phthisis on early ages in the country is

probably due largely to the fact that the greatest amount of aggregation and intercourse takes place in the schools. The relatively high incidence on females at an early age, however, is no doubt due to their greater confinement to the house, and consequently greater degree of exposure, as well as to the higher resistance of boys, who take more exercise and live more in the open air. Are the conditions of school life responsible for the absolutely high phthisis rate at early ages in some counties? The reversal of incidence in males and females in the country as age advances is probably due in no small degree to the influence of inns and public-houses.

The influence of aggregation is, however, not confined to the circumstances which I have mentioned. Wherever a large urban population is collected, there is increase of mutual intercourse and increase of exposure to infection in a variety of ways. The more closely houses are packed, the greater is this social intercourse; the poorer the population, the greater is the degree of crowding. In poor houses lodgers trench on the space available for families, or houses are subdivided amongst families. If houses are crowded on space there is deficiency of light and of cleanliness. At the same time, owing to poverty, there is diminished resistance; hence in the poorest and most crowded districts, such as the Manchester Township and Hulme, we have intensification of the death-rate at each age and encroachment of the disease on advanced age. In poor and less crowded districts, owing to the effects of social intercourse and aggregation in workshops, we have less incidence on advanced age and less severity of the disease, but we have a maximum of incidence on the ages 45—64 in males. This is as well marked in North Manchester as in South Manchester. Through all modifications of age incidence, however, permeates the intensifying influence of poverty.

We thus see that poverty acts in two principal ways: It weakens the resistance to disease, and leads to increased exposure to infection by increasing aggregation and more intimate intercourse both within and external to the dwelling. The prevailing note in the production of the high destitution phthisis-rate in Manchester, and indeed of the high home death-rate, is, I think, the large amount of low-grade labour with the modes of life to which it tends. This appears in various ways.

It will suffice, however, to refer to the classes of labour in which the phthisis death-rate is high. The male death-rates at ages 10 and upwards are given for three years ending with the third quarter of 1903 for a variety of occupations in the "Manchester Health Report for 1906." The total number dealt with is 203,088, with an average death-rate from

phthisis of 3·22. Amongst common labourers the death-rate was 15·5 per 1,000; hawkers, 11; warehouse porters, &c., 12; boot and shoemakers, 6; mechanics, 5·08. These death-rates refer, however, to a comparatively small section of the male working-class population, which cannot be put at more than one-tenth of the whole.

The census of common lodging-houses furnishes the means of knowing to what classes of occupation poverty is attached, and to some extent from what classes it is recruited. Mr. Lock has constructed for a number of years, from the notification records, statistics showing in age groups the classes of workers attacked by phthisis. We may also take the different classes of occupation and measure their liability to destitution by the proportion of the cases notified which are reported from the union hospitals. Adopting this mode of estimating the lower grades of labour, we find that the occupations are for 1903-7 :—

Lower-grade labour	Numbers notified from home	Numbers notified from union hospital	Containing a high amount of lower-grade labour	Numbers notified from home	Numbers notified from union hospital
General labourers ...	153	357	Ironworkers ...	225	162
Hawkers ...	32	172	Warehousemen ...	123	69
Building trade ...	49	90	Woodworkers ...	76	67
Dock labourers ...	9	42	Railway employees ...	40	22
Chemical labourers ...	4	10	Carters ...	48	38
Market porters ...	17	115	Tailors ...	56	27
Warehouse porters ...	19	74	Soldiers ...	40	43
Shoemakers ...	38	47	Barmen ...	54	30
Dyers ...	23	30	Glassworks... ..	17	9
Gasworks employees ...	8	13	Painters ...	18	9
Bakers ...	8	13	French polishers ...	10	7
Coachdrivers ...	12	20	Plumbers ...	11	7
House-painters ...	20	25			
Horsekeepers ...	4	15			

This table reminds us that to every large occupation pursued in Manchester there is a tail of casual workmen, both skilled and unskilled. With this intemperance has no doubt much to do. Had these been deaths instead of notifications the proportion from the union hospitals would certainly have been much higher. A large section of the employed males is represented in the above figures, but the point which it is designed to illustrate—viz., the high amount of labour of a lower grade—is not so clear as it might be, and, in fact, these figures rather suggest that, besides a high amount of casual labour, there is a great deal of poverty due to the wastage from skilled employments. The individuals

thus shed would not be described as labourers in the census. Moreover, the census was taken at a period when unemployment was remarkably low. At such a period many men who would ordinarily rank as labourers would, with some degree of justification, class themselves as skilled workers. Moreover, the ironworker's labourer is liable at any period to classify himself as an ironworker, the warehouse labourer as a warehouseman, and so on. In any case about one-quarter of the population of Manchester may be classed in the aggregate as poor. This estimate corresponds to Mr. Rowntree's estimate of "primary" and "secondary" poverty, and is certainly not over the mark.

Generally the home and union death-rates are highest in those districts which have the highest proportion of the very poor; but, amongst the poor districts, Hulme exhibits figures of an exceptional character, the female and child mortality from tuberculosis being very high. The tables would indicate that in this district the destitute sick are not removed to the union hospital with the same stringency as in the Manchester Township. Out-relief is more readily given. If the differences are to be explained in this manner the result is unfortunate for the children. It is not easy to find another explanation. It is only the home death-rates which are so excessive. Bad as the conditions of housing are, they are not worse than those existing throughout the Township, nor, when the union hospital death-rates other than those in lodging-houses are added to the home death-rates, do they exceed those in the three central districts. There is less crowding, except in Ancoats. These facts, then, suggest that a high degree of segregation amongst the poor does prevent a certain amount of tuberculosis from developing amongst the children of phthisical persons.

It is, indeed, difficult to believe that the removal of so great a mass of infection from the Manchester Township as the tables reveal can do otherwise than influence the amount of phthisis existing in the division. Yet, up to 1908, no material reduction in the death-rate of the Manchester Township was apparent. We have seen, however, that there is no inconsiderable influx of cases of phthisis into this division, and, if nothing more is done than to occlude this quantity, not less than 25 per cent. of the total, a great service will have been rendered to the community as a whole. Still, as a matter of fact, up to 1908 no reduction had been produced in this division. It is in truth a question of production versus destruction of infection. Both are high in the Manchester division. In North Manchester again, in which the amount of segregation has been small, and where it is likely that no more cases are

received into the community than are sent out of it, decided improvement has occurred. South Manchester is intermediate in both respects.

The institutional treatment of disease has increased greatly in Manchester, as in other large centres, in recent years. That it has hitherto exerted no more influence on phthisis is due chiefly to the impoverishment of large sections of the workpeople. As the incidence of phthisis continues to decrease, institutional treatment will produce a greater effect. Whatever reduces poverty will reduce phthisis. No doubt the institutional treatment of disease is in itself a valuable means to this end. It is, however, only one of the means, and it is permissible to hope that other measures will accelerate the pace. Measures of sanitary reform will be passed, and the efficient carrying out of direct precautions will be continued and will continue to produce an effect. A little patience is needful. Benevolence, municipal undertakings, and warehouses continue to attract the misery and disease of industrial Lancashire to the heart of Manchester. There is much wastage from skilled labour. Still, with a little clearer perception of the precise points to which attention should be directed, and a little more effort, we may gradually achieve success. .

No attempt has been made in the above analysis to deal with disease or mortality except in its relation to poverty. The remarkable improvement of recent years in the adult death-rate of Central Manchester from all causes, as distinguished from phthisis, has not come within the scope of this inquiry. In this development the hospitals have, no doubt, taken a considerable part, and to the extent to which they have done so, poverty has been reduced.

PART III.

COMMENTS ON PROPOSALS FOR THE REDUCTION OF POVERTY AND SO OF DISEASE.

At what points can we most effectually interrupt or weaken the vicious circle of cause and effect between poverty and disease? Is it possible to do something more to reduce poverty, and to do so in such a manner as most effectually to influence disease? Or is it possible so to reduce disease as to diminish poverty? We have to consider also what are the consequences which are liable to follow any action taken in either direction, and more especially whether amongst such consequences may not be included the defeat of the object aimed at. It

is this consideration which creates the conservative habit of mind in thoughtful people, and not any lack of desire to advance reforms when these offer an unquestionable prospect of betterment. It is for the same reason that many are averse to interfering with existing institutions and modes of business, further than to amend them, and, where needful, to oil the wheels of the machinery. It is also necessary to consider whether new machinery will work in the manner anticipated by its inventors, and whether the skilled and willing workmen required to work it are in existence and within reach. It is desirable to ask ourselves whether there is any reason why we should not do what we can to amend the existing machinery until we can get new constructed and what repairs are most needed. The following remarks are intended as some answers to these questions. Needless to say, they are largely inspired by the reports of the Poor Law Commission, on which they may be regarded as glosses interposed here and there. Nor do they pretend to do more than touch on some salient points.

I trust that you will not be shocked by the statement of one or two platitudes. As men advance in years they have not that nimbleness of fancy required for the creation of new ideas, and they view them with more criticism. The first platitude on which I venture is, that all ameliorations in the lot of the poor should have as a primary object to enable and oblige the individual to do better for himself.

Whatever benefit is conferred on any class, or on the community through that class, if it is calculated to weaken the individual initiative, will, enterprise, and power of endurance, must be looked upon with suspicion. The goal to which our evolution should tend is that the common workman of to-morrow should have something of the skill and *vis* of the artizan of to-day. The unfit should tend to disappear from the ranks of labour. When we come to think of it, this is what has been occurring for the last forty years, through much suffering and tribulation. The better, more enterprising, and more skilled workmen have increased in numbers relatively to the unskilled. Nor, in spite of jeremiads, has the process been completed. It is largely for this reason, in my judgment, that phthisis and most diseases dependent on physical stamina and nutrition have diminished so greatly, and it is the partial cessation of this relative change which is largely responsible for the retarded rate of the reduction of phthisis.

Is the huge increase in the institutional treatment of disease calculated to weaken the initiative and energy of the poor? Under certain conditions I do not think that it is. The first condition is that

the medical service is effective. As Dr. McVail has shown, this can only be attained in all rural unions by combination. The curative treatment of illness is, however, only part of the treatment of disease: prevention is not only better than cure—it is often a necessary part of cure. There seems to be no reason why training in personal habits should not be given to persons in union hospitals who are suffering from chronic chest disease, materials being supplied to them and care exercised to see that these materials are used. It might be tried.

In Poor Law nurseries there should be some one competent to give to mothers instruction in the care of their infants, and whose duty it should be to do so. But, in fact, it is probable that this important side of treatment is better provided for in many Poor Law hospitals than at voluntary hospitals. It is a grave evil that the out-patient department of voluntary institutions and dispensaries in general should be crowded with diseases of all sorts, many of them infectious. No doubt precautions are occasionally taken. It is doubtful whether they are sufficient. This evil is greatest at hospitals for children and dispensaries in general. How best to meet it one does not see. To some extent it is met by the appointment of out-patient visiting medical officers, who exercise a selection of suitable cases for the hospital and out-patient department. Possibly this system might be developed. It is, of course, a question of expense. Another way in which it might be met would be to appoint all medical practitioners officers of one or other of the voluntary hospitals, and to require a medical certificate from a practitioner with every child sent to a dispensary, stating that the case was a suitable one to be taken to the out-patient department or dispensary. By this means a considerable check would be imposed on the abuse of hospitals, while infectious cases of a defined character would be kept away.

The treatment of cases at out-patient departments and dispensaries is of necessity at high pressure, and is not to any material extent preventive. Yet it cannot be doubted that more good might be effected in most cases by careful instruction in the management of children than is effected by the medicine and brief directions given. If this department of work were handed over to another medical officer to carry out in appropriate cases, after the present kind of consultation is finished, much benefit would accrue. More care should be bestowed on this department of medical teaching, which might form a branch of the public-health teaching of our great medical schools. This kind of medical treatment is at present given by many practitioners, and might

usefully be extended to many institution cases, preferably by means of visiting officers. As they stand most in need of it, the poor should receive most careful consideration, treatment, and advice, nor can this requirement be met by a system of provident dispensaries. For all such work they should pay, as for all public assistance rendered. But money payment is out of the question with most of them. It becomes necessary to have a standard of earnings and available income above which money payment should be made, graduated so as not to press severely on the family. An inquiry department is thus required, to which cases should be referred. It should, I think, be obligatory on public charities to refer all their cases to one such department. A public charity is not on the same footing as a private individual. There does not seem any sufficient reason why the same inquiry department should not serve for Government aid as for charitable aid, and there would be great advantage in this arrangement. The sums exacted would not, however, pay for the assistance rendered, and, in addition, further payment should be obtained to the extent possible. The only conceivable manner in which persons unable to pay in money might pay in kind would be by compliance with sanitary requirements. To some extent the public would receive back in this form the value of the assistance rendered. It is suggested, therefore, as a condition of public medical assistance; that the persons assisted be required to comply with sanitary requirements, and that any default be reported to the Public Health Office. Such reports are practically never received at present. These observations are not to be taken as reflecting on our charitable hospitals, which have conferred an immense benefit on the nation, and to which, probably, are due no small share in the reduction both of disease and of poverty which this generation has witnessed.

It would be of very great value to many young medical graduates if the material contained in the union hospitals were open to them and if it were possible for the chief medical officer in these institutions to give post-graduate demonstrations. This class of work is just what is required to supplement the knowledge of disease acquired at the general hospitals, and to give to medical men entering on practice a better balanced view of the work which they are undertaking.

It will be evident from my previous observations that, in my opinion, were it only to obtain more complete views of the tendency of public-health work, it is necessary for men engaged in the public-health service to take some interest in labour problems. The problem which is most urgent is the decasualization of labour. Whether it will be

possible so to organize labour exchanges as to contribute materially to this end remains to be seen. There are at least four functions of a different character which are to be handed over to these bodies:—

(1) By intercommunication between exchanges to fit the disengaged skilled workman with work.

(2) To abridge the intervals of employment of gangs of navvies and labourers engaged on public works, and to conduct them with a minimum of loss of efficiency and effort from one job to another; let it be added, with as little exposure as possible to infectious disease.

(3) To facilitate the transition from one employment to another of workers affected by the seasonality of their work.

(4) To so organize casual labour in individual centres as to keep the same men employed continuously, or as nearly so as possible.

It is the last piece of work which is most urgently needed, but in the way of which the difficulties are greatest. Each employer will wish to keep his own men as far as possible, and with many of the men themselves difficulties will arise. Yet this is an organization of labour which would be practical, effective, and greatly to the benefit of all concerned. It will, however, require much ability of various kinds in the local exchange officers, and it is to be hoped that very great care will be exercised in the choice of men for these important positions. It is sometimes assumed that the work to be done can be so arranged that men can be continuously employed, while the residuum thus created might be provided for by Government schemes, which could be enlarged or contracted according to the conditions of trade. To some extent events might be moulded in this direction by an able and tactful organizer. But it remains that a large section of the casual work would fall on particular parts of the day, and the whole amount of work available, even if it is continued over six days of the week, will be insufficient. Such is much of the labour about markets. Still, even this reform would be to the good. The statement of this part of the problem enables us to understand the *rationale* of the common lodging-house. There are many men living in our common lodging-houses who earn a small sum in the markets for perhaps two or three days of the week; enough, however, for their meagre requirements, which will not exceed 1s. to 1s. 6d. per day. The work of the more capable men, however, might be regularized and distributed among the markets and warehouses. If this were found possible, it might be made a condition that the selected men should live either in a recognized lodging-house or in a private house. There are, or were in 1899 and 1902-3, living in common lodging-houses at 4d. a night some men in

receipt of good wages. This should, in my opinion, not be permitted. In view of the extent to which disease appears to be produced in common lodging-houses, with their close aggregation of men, these should be subject to stringent conditions as regards structure and administration, care being exercised to include all the places which come under the definition of common lodging-houses.

Only when labour has been organized and provision of supplementary work made by Government will it be possible to proceed to the establishment of labour and detention colonies.

Is it possible to provide against trade cycles or trade cyclones, as they have been called? They can apparently be foreseen. In periods of general depression the machinery of labour exchanges will be unavailing to avert distress. Professor Chapman suggests that production might be curtailed during the trade boom. But this could only be effected by international arrangement, as the producers of one country would otherwise fear to be permanently ousted by the producers of another. To effect this object trades unions are invited by him to consider whether greater elasticity of wages might not be permitted, higher wages being required in prosperous times and lower in adverse periods. In this way production would tend to be levelled. This plan might, however, be broken down by foreign competitors introducing new classes of goods when demand was brisk. The suggestion may lead to something, though it is unlikely that it has failed to occur to business men.

Can these cyclones be met by voluntary insurance against employment? Partially, no doubt, this is possible, and, in fact, such insurance is said to be extending. But it is hardly possible for the lower ranks of labour, and the wastage of labour is enormous. No doubt, as the operation of the Workmen's Compensation Act comes to be better understood, it will operate in favour of older men. But the general trend is otherwise so far as unskilled work is concerned. Older men are here, in all probability, replaced rapidly by those who are younger and more active. The immediate occasions of displacement in normal times are often, no doubt, drink and illness. But when the pinch of bad trade comes there is a tendency to displace older men. It is easy to see why there are so many of the older men in destitution, so many men suffering from sickness, and so many relatively intemperate. Many of these men are skilled artisans and will not make good labourers. Labour has its own acquired skill. Neither by insurance nor labour exchanges can this rapid wastage be met. On the other hand, we have the ranks of casual

labour recruited by youths of 18 discharged from some employment which leads nowhere—messengers, shop-boys, and so forth. In one way relief would be afforded if these youths could be educated in such a manner as to be capable of entering the phalanx of the skilled artisans. There is naturally not much enthusiasm in the matter amongst artisans. But it is probable that the pressure of a considerable body of trained youths would facilitate and induce the formation of new industrial enterprises, and would not affect unfavourably present workers to the effect anticipated. There would also necessarily be an improvement in the quality of the higher kinds of work, as well as in the lower.

How the training is to be given is not at once evident. We will assume, however, that such instruction has been organized and is general, partly in works, partly in workrooms under the Board of Education or Education Committees. It is generally conceded that relief works are very wasteful. If, however, the appropriate labour were engaged for such works, the surplus would partly find its way into employment, and partly it might be dealt with as follows: Selected unemployed labourers to be put under a regular course of training in workshops, with a minimal allowance for maintenance, on certain conditions. The unemployed skilled workers to be utilized as teachers, of course under direction. This proposal might involve the partial scattering of both one and the other, but with a system of labour exchanges established this defect could easily be remedied. This proposal for the absorption of labour would probably be ultimately less expensive than the provision of relief works, would have the advantage of keeping the persons provided for continuously occupied, and, while raising the whole status of labour, would provide against the next trade boom a useful potential supply of partially skilled workmen. In any case, the mechanical skill acquired would be a source of interest in household matters, and would be of frequent use to the persons instructed. Amongst other outlets which such training would provide might be the making of ingenious toys and games.

There is, indeed, a feeling of unrest in regard to education. While it is admitted that the power of the present generation of adults has been greatly extended in respect of literary enjoyments and pursuits, there is no such crop of literature as one would have expected, nor do the habits of the younger people seem to have improved to the extent which one might have anticipated. The fact is that unemployment is a blight which devours the fruits of progress. But, in addition, while teachers have been kept at relatively high tension, and the higher class

of teacher is more accomplished and skilled in imparting knowledge than the teacher of the past, owing to the scale on which their work has been carried on, their abilities and skill have been used in imparting knowledge instead of being expended in stimulating work and guiding study. Their work is thus, to an undue extent, wasted. It is not unmastered and undigested food which nourishes the mind any more than the body. Education in any true sense means that the child has been taught to perceive and overcome his difficulties by the output of his own effort. If, through the skill of the teacher, knowledge is temporarily acquired without serious exertion, it is not a permanent possession, but is lost at the first contact with the life of toil which exacts effort and confers faculty, however limited. There is, however, a more serious evil resulting from the absence of individual and independent exertion. The whole habit has to be acquired later on, and in too many situations there is little opportunity for acquiring it. In general there is a serious loss of initiative in the young adult.

Here, again, I must be content to point out what I believe to be a defect. It can only be remedied, I fear, at a great additional cost in the machinery of education, and it is, no doubt, the prospect of such expense which has delayed progress.

It is, again, a matter of the most serious moment that girls should be taught all that pertains to the management of a home. The homes of the working classes are at present very depressing. Whether such knowledge is imparted through extension of the years of schooling, or subsequently in continuation classes, is not, perhaps, of the first order of importance, though of that I am not certain. At all events, it is vital that such training should be given. It should include education in the choice of a house, in habits of order and neatness, the essentials of cleanliness, the principles of dieting, the selection and purchase of cheap and wholesome foods, the keeping of accounts, the cooking of foods, especially of the cheaper kinds, so as to vary and appetize them, the selection of stuffs, needlework, mending, and, above all, in the rearing of children.

Boys also should be instructed in the construction of cheap diets, in a knowledge of foods, and in the elements of personal hygiene. The loss of health and means incurred by ignorance and thriftlessness, the consequence of our industrial system, is enormous. I must avow my belief also that it is chiefly by instruction in regard to the proved effects of alcohol, economical and physical, that a very great influence is likely to be exerted on the working classes. The results can only be gradual,

and must be based on the plainest and most indisputable evidence and reasoning. Such instruction might well come under dietetic instruction.

Here, again, we are encountered by the spectre of expense. The instruction for which one pleads is, however, to my thinking, a matter of life and death, and could not fail to repay its cost with interest.

In the improvement of the health of children a great step forward has been made by the medical examination of school children. We have followed the track marked out by the Germans and the Americans, and the system has been started with the energy which will always, we may hope, characterize our people. In my judgment there is, also, urgent need to train all aspirants to the teaching profession how to observe in children the signs of disease, whether mental, infectious, constitutional, or belonging to the separate senses. Such instruction must be practical, but need not be very deep, since the functions contemplated on the part of teachers are intelligent observation of the state of health of the child and good scouting, if the expression may be used.

Their opportunities of observing each child are numerous and continuous. But it is axiomatic that most people observe very little of what is passing before their eyes unless they are trained to do so. What the teacher requires is training in the observation of the child's state of health. It is true a little intermittent training is given when the medical examinations are made, but that does not amount to much. If, however, the teacher were trained to note signs of illness, and if, in addition, periodic determinations and records were made in every school of the height and weight of the children, the gain to the system of medical inspection would be very great. The difficulty here is, of course, the resistance offered by the teachers to such an extension of their training. But, on the other hand, their usefulness to the nation would be correspondingly raised.

The value to be attached to records of height and weight is becoming better recognized. In this connexion I should like to refer to the important observations of Miss B. Walton Evans in regard to boarding out of pauper children (Annual Report of the Local Government Board, 1908-09).

With the Board of Education more than any other body rests, I am convinced, the future of the working classes in this country. If one appears to be advocating a great addition to the cost of education, it is in the firm conviction that along the line advocated lies the path of true economy.

One suggestion for relieving the pressure of trade depressions has

been made. A still simpler method would be to vary the period at which education is to terminate, power being conferred on the Secretary of State to alter such period from time to time according as the necessities of the nation appear to demand.

In the minority report the extension of the age at which compulsory education should cease to 18 is advocated. So great an extension is, possibly, more than the back could bear, under present conditions. But the age might be advanced to 16, with power to vary it in the forward direction from time to time. Such a power would provide a considerable relief to the labour market. It would be limited, as to the period of extension, by the necessity of absorbing into any organization established for the special kind of education contemplated the additional scholars set free. Another variant of the same proposal would be to advance the male age to 15, the female to 17, with power to vary.

We have seen, however, that there is a large section of incurable loafers, incapables, and degenerates who would ultimately find their way to detention colonies, many of them by way of labour colonies. There can be no doubt that such colonies are a necessity of any real advance, and with the principle of detention, and, for a section, of permanent detention, one cannot but be in full sympathy. Again the expense looms large, but undoubtedly this expenditure would be economical in the long run, if wisely made. The same may be said also of Miss Mary Dendy's work in the establishment of detention homes for feeble-minded children, which she has rendered permanent in character by her great ability in administration. Indeed, it would be difficult to overrate the increase in efficiency of education and the prevention of suffering, demoralization, and inefficiency which this system, extended as no doubt it gradually will be, is capable of effecting.

But the waste of our industrial organization is immense, nor is it likely readily to diminish. Why do men drink? Chiefly to obtain a respite from care and fatigue. If a man is out of work he drinks because he is down on his luck, and his mates are willing to help him to forget. But if he is in luck and overworked, he drinks to escape the sense of fatigue. On the other hand, if relays of men are employed for pressure jobs, casual labour is created. It is difficult to see how men and women are to be freed from this tyranny without some such proposal as that of the minority report in regard to education being carried into effect, and with the aid of effective labour exchanges. One reservoir of labour would then be found in the local labour exchange, and, failing this, the supply would be obtained from young men undergoing instruction.

The essential thing is that men should not be idle. But it is little less essential that they should not be overworked. No provision is made under any of the proposals for the prevention of that peculiarly tempting form of idleness which consists in doing a little work, just enough to maintain a man in a common lodging-house, with his requisite allowance of cheap liquor. There is no form of loafing more dangerous to the individual or to the community, whether from the point of view of efficiency or disease. There appears to me to be a real danger that, in proportion as the organized effort of the community is successful in providing continuity of labour, the large class who do not want continuity of labour will more and more elude reform by gathering into these places, in which disease abounds and economy is next to impossible. They should be subject, I think, to stringent rules, and it is worth consideration whether men in receipt of an income available for their own needs exceeding sixteen shillings a week, and young men, should not be altogether excluded from them. There should certainly be power to detain in the union hospitals men suffering from phthisis admitted from common lodging-houses or from private homes when, in the opinion of the medical attendant admitting them, their restoration to liberty would endanger the health of others, whether of other lodgers or of their own households. This reform is an urgent one, and should not await the decision as to who is to be the authority for administering Public Assistance (or Poor Law) Hospitals.

Amongst all the reforms which are advocated I would, however, place the greatest stress on the reforms of education, and among these on the additional education in manipulative skill and in domestic management. Great, however, as would be the influence of these, it is as with labour : human nature would assert itself, and we should still have many unthrifty and slovenly homes and many neglected and injured children. For these the Notification of Births Act and the Children Act provide a possibility of government. It is, however, conditional on the creation of a staff of well-trained and suitably paid health visitors, and it may be necessary in course of time to make obligatory the provision of such a staff in all districts fulfilling certain conditions to be defined by the Local Government Board.

I have not ventured to deal with the question of public assistance, its mode and measure. In regard to the great scourge of phthisis, indeed, I have urged that adequate assistance should be afforded to the families invaded by that disease to maintain the other members at a reasonable level of nutrition. This would be given, however, only with due safeguard

in the way of removal and detention of the phthisical member of the household, when, in the opinion of the responsible authority, such a measure is required, and conditional also on good security that the assistance given is not wasted. Where no such security could be obtained sufficient to ensure the proper use of public assistance, it would be necessary to take steps to protect the public against infection. There is general consent that, at the present time, a considerable section of the population is living in a manner calculated to produce a debile adult race, and that the relief afforded is often insufficient to avert this result.

On the other hand, it is clear that there is great danger if adequate assistance is given that incapacity will be endowed at the expense and to the disablement of capacity. Under the proposals of the minority Commissioners mothers in charge of children are to be required to fulfil their proper function, and are to be prevented from going out to work. Adequate assistance is to be given where the families are large, so that all children may be sufficiently nourished and cared for until they become self-supporting. I have, with others, maintained that infants and children should not be allowed to suffer through malnutrition. Adequate physical training for children is generally recognized as a necessity, which in turn entails the necessity of adequate nourishment. This principle, however, holds good not merely for the present, but for the future, and, if adopted without safeguards, must result in the fostering of incapacity and the degradation of the national standard. Its recognition entails other principles without the application of which it must add to existing evils. It should be unlawful to unite in marriage, by any of the recognized methods, any persons who cannot show some immediate provision, besides the possession of assured and sufficient means for the future or tenure by the intended husband of employment at a suitable wage with reasonable prospect of continuance. A certificate from the medical practitioner who has had the largest experience of each, that each of the two is free from any disease which would be calculated to injure the offspring, should also be required. Some means might have to be devised for rendering illegitimacy a source of considerable trouble to both parents, while not affecting the children.

It does not appear just that so much should be done for the destitute at the expense of those who just avoid destitution, without return. On the other hand, it does not appear desirable to start a number of young men and women in life with a load of debt round their neck. The debt to the State might be defrayed in various ways. In the first place, the State might have a lien on the services of the young men and women

whom it had supported or trained, and might exact in return a period of service in one or other capacity ; or one of the workers being placed *in loco parentis* to the rest of the family, his services in that capacity might be recognized as payment of his debt ; or finally, if he so preferred, he might, on the security of friends, be permitted gradually to discharge his debt by payment. The subject is, however, difficult, and not one for dogmatism ; nevertheless, it is most desirable that every man and woman should practically feel the need of independence.

In conclusion, I must express my deep sense of inadequacy to the task which I have undertaken. There is great need for increased individual efficiency ; to this end the poorer classes must be trained, organized, and assisted ; but at the same time the sense of independence and the individual responsibility must be maintained.

On the whole, we have advanced in this direction, but the dangers ahead are not slight, nor have we approached the development of which the nation is capable. The boldness of the measures advocated in both reports of the Poor Law Commissioners is proof that a strenuous effort will be made to arrest the creation of poverty, and thereby to diminish disease ; and also more directly to diminish disease as one of the main sources of poverty. May the measures taken be effectual without creating fresh difficulties.

Epidemiological Section.

November 26, 1909.

Dr. JAMES NIVEN, President of the Section, in the Chair.

The Case-incidence in Nine Epidemics of Measles at a Public School, with Notes on the Pre-eruptive Symptoms.

By H. G. ARMSTRONG.

ASSOCIATION with a large school or similar institution gives its medical officer the opportunity of seeing the common infectious diseases under conditions somewhat different from the general medical practitioner. I propose in this paper to give some of the results gained from the experience of the various outbreaks of measles which have occurred at such an institution during the period of twenty-five years that I have been in medical charge of it. The commencement of it coincides with the first publication of the "Code of Rules for the Prevention of Infectious and Contagious Diseases in Schools," which has since been in common use by the medical and other officials of most public and private schools. This was shortly followed by a paper on the "Liability to Infection during School Life, and its Relationship to Sanatorium Accommodation in Schools," by C. E. Shelly, then medical officer to Haileybury College.¹ In it he showed—using the simile of a box of cartridges, some loaded (the unprotected), some unloaded (the protected)—that an epidemic of measles might be expected in a school whenever the total number of the unprotected boys equals one-third of the total number in the school—i.e., when the susceptible material is so concentrated as to form a readily explosible mass.

The school, when I first became attached to it in 1884, contained 400 boys, which, with some fluctuations, has been increased to 520. The total number of boys admitted during the period has been 3,260 ;

¹ *Practitioner*, Lond., 1890, xliv, p. 461.

the annual rate of admission has varied from about 130 to 150. The average age at entry is $13\frac{1}{2}$ years, and the average time that each boy remains in the school is about $3\frac{1}{2}$ years.

In a school of this class, where the average age at entry is 13 to 14 years, it was shown by Shelly in 1890 that 25 per cent. of the entrants were unprotected by measles; in those that I furnished him from my school for his statistical inquiry, the figures stood at 24 per cent. unprotected as against 76 per cent. protected by a previous attack. The dissemination of the knowledge of prophylaxis provided in the "Code" amongst the public and those in charge of preparatory schools, where quarantine precautions are more easily carried out, has caused a gradual increase of the percentage of the unprotected, who reach the age of entry to a public school, and in my case the figures now stand at 30 per cent. unprotected and 70 per cent. protected. Of the 3,260 boys who have entered the school during the twenty-five years, 2,380, or 73 per cent., were protected, and 880, or 27 per cent., were unprotected. Of these, 617 were attacked at school, leaving 263, or 8 per cent. of the whole number, who escaped; but this does not take into account cases which may have occurred in the holidays.

The disease has been introduced into the school on twelve separate occasions. On three of these it did not spread, these abortive epidemics accounting for five cases. The remaining 612 cases were divided amongst nine epidemics; seven of these occurred in the spring terms and two in the summer. Each outbreak has been complete—that is to say, practically all the susceptibles have been attacked.

I have prepared a chart showing the case-incidence in each of these nine epidemics. An examination of this shows that there is an almost definite rule that the larger the epidemic the quicker it is over. Thus in 1898 an outbreak of 118 cases was completed in 32 days, while in 1900 an outbreak of 53 cases took 51 days. The reason for this will, of course, be found in the fact that the greater concentration of the unexploded material leads to a more rapid and complete combustion. The ratio, between the date on which the 10 line is reached and that on which the epidemic is completed, is almost in proportion. Thus there were in—

1898	...	118 cases in 32 days	...	10 being reached in 10 days
1887	...	101 " 37 "	...	" " " 13 "
1904	...	99 " 40 "	...	" " " 12 "
1907	...	76 " 60 "	...	" " " 16 "
1900	...	53 " 51 "	...	" " " 20 "
1909	...	46 " 37 "	...	" " " 16 "
1891	...	43 " 33 "	...	" " " 15 "
1908	...	43 " 40 "	...	" " " 13 "
1893	...	33 " 38 "	...	" " " 23 "

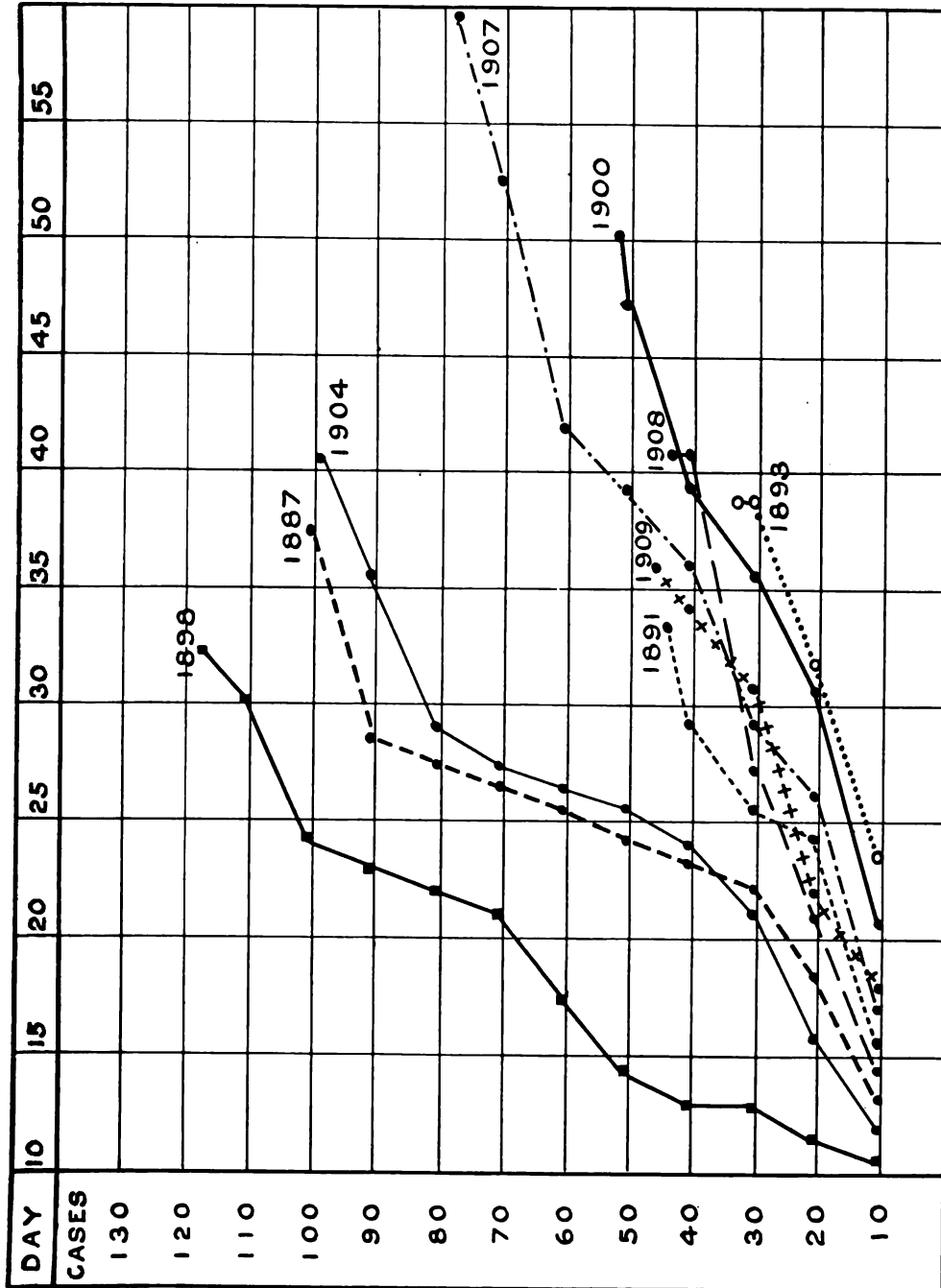


CHART I. Case Incidence in Nine Epidemics of Measles.

The epidemic of 1907 seems not to support the rate of incidence indicated by the other outbreaks. But there were special local circumstances accounting for the difference. This epidemic occurred in a summer term, in which there were urgent reasons why it was of the greatest importance to prevent its spread. With this in view, all the susceptible boys who were known to have been associated with the original infector were kept under rigid observation, with the result that 13 of those infected were isolated before they had themselves become infectious. But one boy, who was not on the suspect list, developed the rash on the seventeenth day from infection, and thus restarted the epidemic. Therefore, deducting the original infector and 13 of his victims from the whole number and 17 from the number of days, the figures should read 62 cases in 43 days, which brings this epidemic into line with the others. This table may chiefly be of value as a means of indicating to the authorities of an institution the amount of hospital accommodation that is likely to be required. The larger epidemics, owing to the greater rapidity of their spread, will require a considerably greater proportion of beds than a smaller one. Thus in the 1898 epidemic—giving 21 days as the duration of each case—out of the 118 cases, 108 were in hospital at the same time. In 1887, with 101 cases, not more than 70 beds would be required, and for the 53 cases in 1900 the incidence is so slow that 30 would suffice.

On the introduction of a case of measles into the school, after estimating the probable size of the outbreak, which will be 30 per cent. of the entrants since the last epidemic, I make out a probability table of the maximum number which will be under treatment at any given time, and the necessary accommodation is provided. In my first epidemic, for the want of such knowledge of probabilities, I found myself one evening with every available bed occupied; and it was only by strenuous exertion and keeping workmen employed through the night that provision was made for the next day's entry. As an illustration of the value and comparative accuracy of the table I may mention the following instance: The medical officer of another public school, writing to me, said that a case of measles had been introduced and that he expected a big outbreak, as there were 170 unprotected boys. I sent him a copy of this table, with the prophecy that on a certain date he would have 150 cases. He informed me afterwards that the actual number on the date mentioned was 153.

Measles being infectious during the pre-eruptive stage, and the avenues of possible infection being so many, there is little probability

either of preventing the introduction of the disease into a community or of limiting its spread after it has been introduced. As I have already shown, amongst boys of the upper and middle classes attending secondary schools, 70 per cent. have been attacked before reaching the age of 13 to 14 years. The proportion is probably much larger among children attending the elementary schools. From a census I took last month of the children attending the elementary schools in my rural district I found that the proportion of those attacked was 76 per cent. of the whole number, and of those who had reached the age of 10 years 87 per cent. Dr. Sinclair, the medical officer to the Post Office, has furnished me with the information that amongst the telegraph messengers in the City area 78 per cent. had the disease before joining the service. If, however, any success is to follow an effort to arrest the progress of an epidemic, it can only be by recognizing the phenomena associated with infection and the symptoms of the early or pre-eruptive stages, and so making diagnosis and isolation possible before the infectious stage is reached. *Infection* seems always to be due to direct personal contact. I have found no evidence that it is ever conveyed by fomites or by an intermediate carrier. The negative evidence of this is very strong. There is employed in the school a large number of masters and servants living in their own homes. But no instance has occurred in which these have conveyed the disease to their families, nor, on the other hand, in which it has been conveyed from the family to the school. The poison is air-borne to a limited distance. Observations made in the cases where infection has spread during the time of attendance in chapel indicate that the outside range is 9 ft., but the great majority of the infected were found to be within 6 ft. of the focus. The 6-ft. range prevails in the winter, but in the summer, when open windows and doors cause a more rapid movement of the air, it may be increased to a 9-ft. radius. In two of the epidemics almost the precise moment that the infector commenced to distribute the poison could be traced. In one of these it was 17 hours, in the other 24 hours, before the appearance of his rash.

Incubation Period.—The only practical way of determining this is by calculating from rash to rash. The commencement of the prodromata is too irregular and intangible to allow of accurate recognition. In the nine epidemics the original infector in each accounted for 160 cases altogether. In the accompanying chart I have set out the number of days from probable exposure on which each of these respectively was diagnosed, together with a percentage table of the

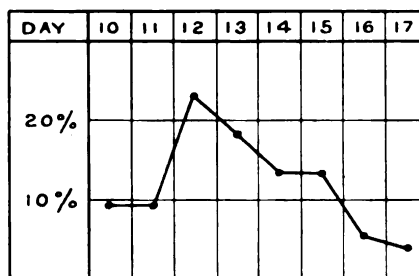
50 Armstrong: *Case-incidence in Epidemics of Measles*

whole number. From this it will be seen that 74 per cent. occurred by the fourteenth day.

Immunity.—The protective influence of an attack is nearly complete. Of the 617 cases, only 2 per cent. were reported to have had previous attacks, on evidence which on investigation seemed to be satisfactory. It has not been my experience to attend any patient with two attacks of the disease.

DAY	10	11	12	13	14	15	16	17
1887	1	2	2	6	1	1		
1891				4	1	4	5	1
1894					1	2	1	1
1898	10	6	19	10	6	5		
1900					1	1	2	2
1904	2		4	3	8	5		1
1907		1	2	3	2	3		2
1908	2	7	5		2	1	2	
1909	1	1	3	3			1	1
TOTAL	16	17	35	29	22	22	11	8

Table of Incubation Periods.



Percentage Table of above.

CHART II.

Prodromal Symptoms.—The earliest of these, probably, is the alteration of weight, known as Meunier's sign. He says: ¹ "There exists during the phase called incubation of measles a phenomenon which we have constantly observed, and which consists in a marked lowering of

¹ *Gaz. Hebd. de Med. et de Chir., Par., 1898, N.S., iii, p. 1057.*

body-weight, independent of every kind of morbid troubles—digestive, secretory, or other. It begins about the fourth or fifth day after contagion—that is to say, five or six days before the appearance of the first catarrhal or febrile symptoms, eight or ten days before the eruption. It lasts several days, more often even to the beginning of invasion. Its intensity varies with the case, but seems independent of the age of the subject and of the severity of the later measles. The loss of weight is about 10 oz. to 1½ oz. in a child of one to four years; it may reach

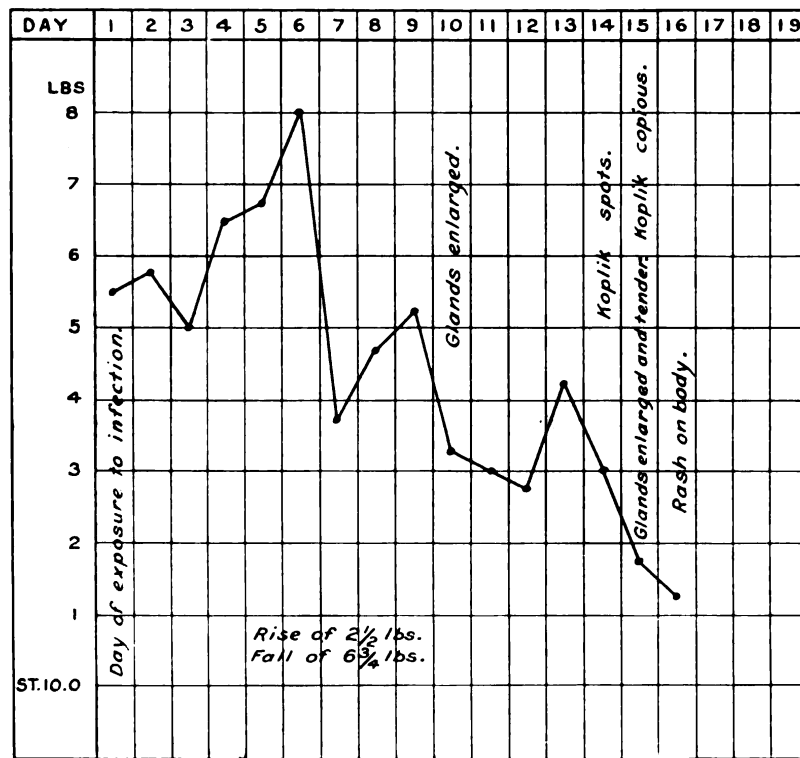


CHART III.

22 oz. and has not been observed less than 3 oz.” I have not been able to find that since that date, any observations have been published confirming or otherwise those of Meunier. I have myself made a considerable number, and am showing to-night four charts which are selected as typical. In each the same phenomenon exists—i.e., a preliminary rise up to the fifth or sixth day from contagion, followed by a fall which lasted up to the day of invasion. The fall is much greater than that

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indicated by Meunier's, and probably bears some proportion to the weight of the individual.

Chart		Initial weight		Rise of		Fall of
III	...	10 st. 5½ lb.	...	2½ lb.	...	6¾ lb.
IV	...	8 st. 3¾ lb.	...	1¾ lb.	...	5 lb.
V	...	8 st. 10½ lb.	...	1¾ lb.	...	3½ lb.
VI	...	5 st. 4½ lb.	...	2 lb.	...	3 lb.

In connexion with this alteration of weight, it is interesting to compare the study of the blood in measles reported by Renaud and

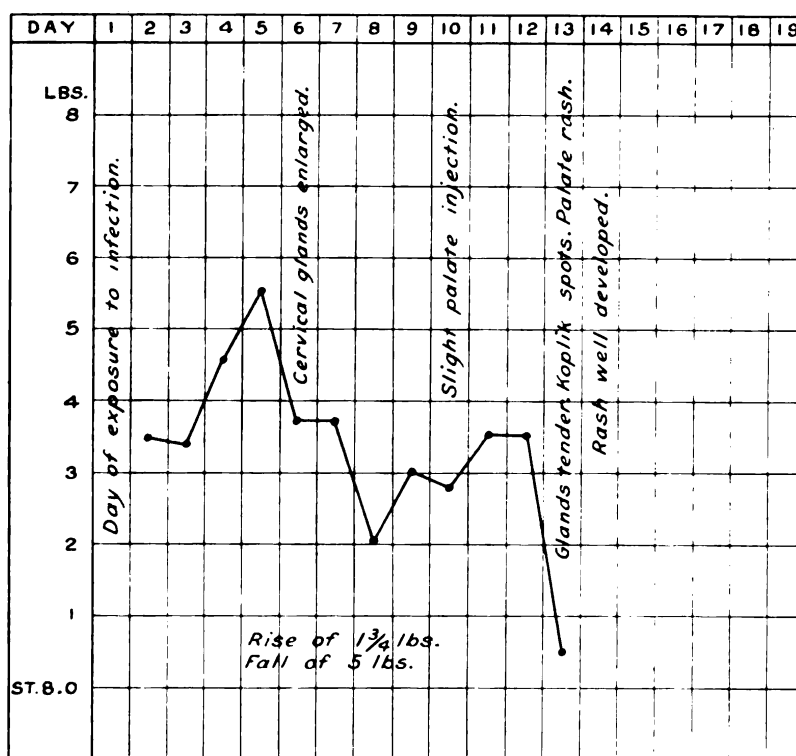


CHART IV.

Tileston respectively. They found that there is a leucocytosis beginning early in the period of incubation, reaching its maximum six days before the appearance of the eruption and lasting into the first part of the stage of invasion. During the latter part of the stage of invasion the leucocytes fall to normal. The curve of the rise and fall of the weight and that of the leucocytes seem to be identical.

Lymphatic System.—From the sixth to the eighth day from contagion, coincident with the commencement of the decline of the body-weight and of the leucocytes, the superficial glands, especially those in the cervical and axillary regions, become enlarged; not at first tender, but generally becoming so a day or two later. This implication of the lymphatic system is found, of course, in all the infectious disorders, and markedly so in rubella. But in rubella the implicated glands are more numerous, harder, and more shot-like; those in the mastoid

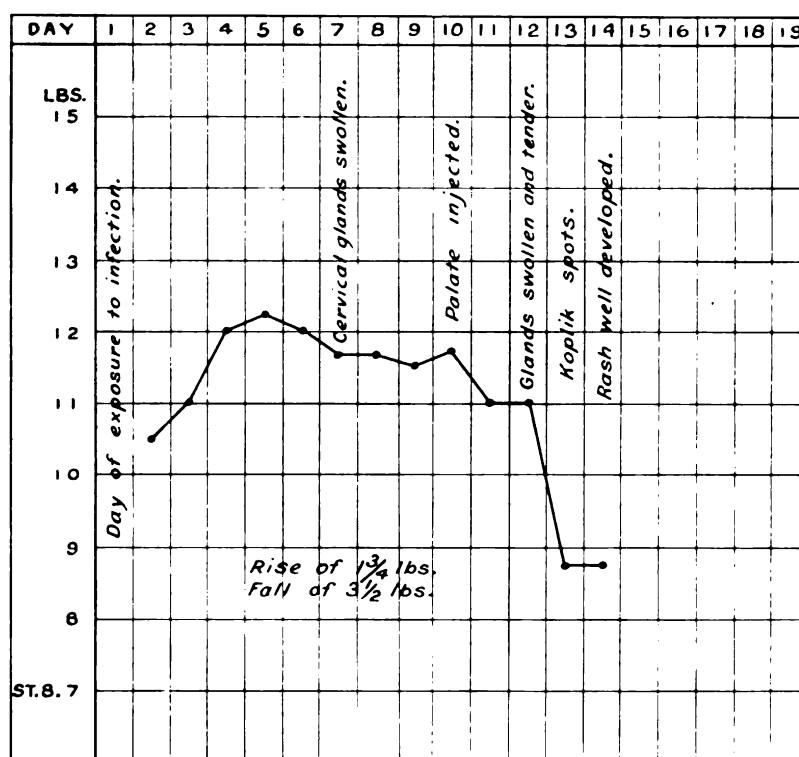


CHART V.

and sub-occipital regions, which seem to escape in measles, being always affected.

Prodromal rashes have very frequently been observed. These have nearly always been of an urticarial type, sometimes appearing shortly after contagion, but at any time during the initial stage. The erythematous eruption, simulating scarlet fever, which is described by several observers, has not been seen in any case. I have very frequently seen a

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slight and transient macular eruption on the skin of the chest in the region of the nipples appear at the commencement of the stage of invasion, but disappearing completely before the appearance of the typical rash.

Koplik's Spots.—During the last four epidemics I have made a constant practice of looking for these and have almost invariably found them present. The day of their appearance is, however, variable; sometimes they were seen several days and sometimes only a few hours

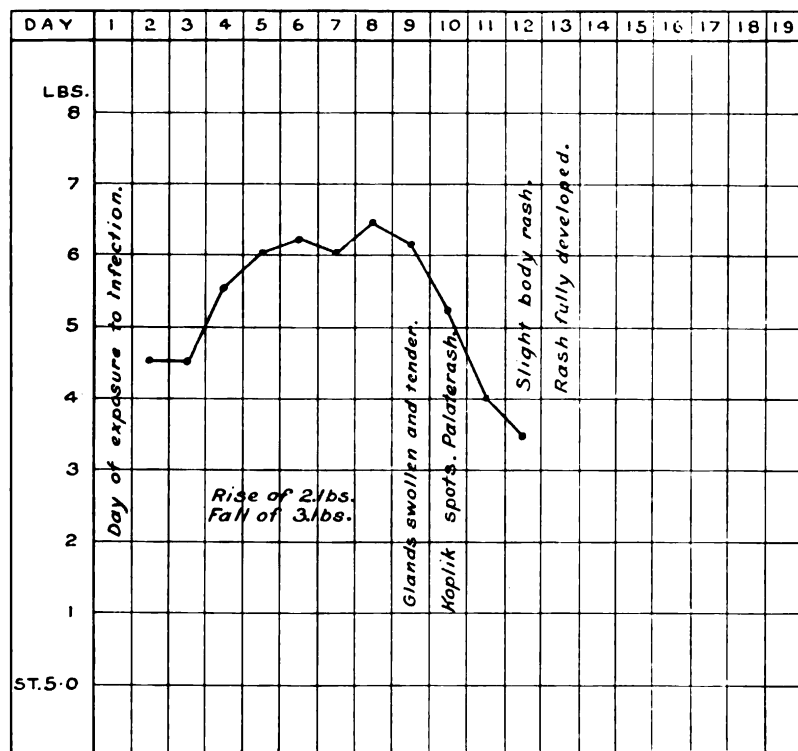


CHART VI.

before the general exanthem. From the point of view of a pre-eruptive diagnosis their presence may be regarded as a positive sign, but their absence must not be taken as a negative one. Coincident with the spots on the buccal mucous membrane, and sometimes preceding them, there is a peculiar mottling of the soft palate with a congestion of the veins at the margin of the anterior pillars of the fauces.

From eight to eleven days after contagion the well-known symptoms

of the catarrhal stage commence: drowsiness, coryza, sneezing, malaise, and some fever of an indefinite and variable intensity. The peculiar odour of measles, which was likened by Heim to that of freshly-plucked goose-feathers, is very perceptible in a ward full of patients. It may occasionally be detected in the early stages before the appearance of the rash by a sensitive and educated nose. There is frequently a remission of all the symptoms for a short time immediately preceding eruption, more especially as regards the temperature, which often falls to normal. In Chart VII are given two pre-eruptive temperatures, which are fairly typical of those which may be met with.

Observation of suspects on these principles makes it possible to arrest an epidemic in its very earliest stages. On three occasions, with the school in a highly explosive condition, I have succeeded in doing so. In

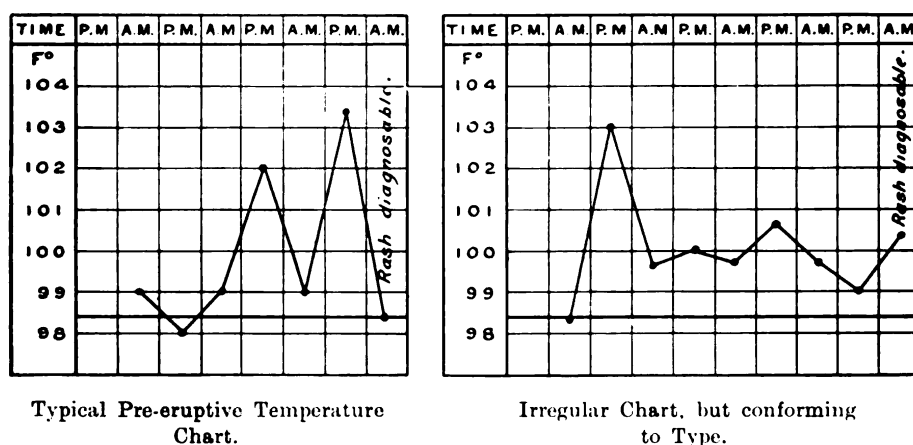


CHART VII.

another I nearly did so, and only failed in consequence of an unmarked suspect not having been kept under observation. Experience, however, shows that no real value results from the attempt to limit the progress of an epidemic. In spite of the closure of elementary schools and other quarantine regulations, practically every individual is attacked sooner or later, and no result commensurate with the expense and trouble given has followed. From the figures given above it will be seen that, of the children of the upper and middle classes attending secondary schools, 70 per cent. have been attacked by the age of 13; and of those attending elementary schools, 87 per cent. by the age of 10. And in this the latter have the advantage, as an attack is more liable to be severe in the

later years of adolescence than the earlier. I know it will be urged that the mortality from measles is very great; but the same remark applies with equal, if not greater, force to whooping-cough. But it is as hopeless to stop the spread of one disease as the other. Should any success attend our efforts to limit the progress of any particular outbreak in a community, it only results in swelling the number of the susceptibles to be attacked in the next. I believe that the principal danger of measles lies in the size of the epidemics, and that there is a greater proportional case-mortality in large than in small ones. I have always been struck with the increase in the severity of the cases each epidemic presents as it progresses. The mortality has not been large, but, so far as it goes, illustrates this point. There have been 5 deaths, or 0·8 per cent. of the whole number, occurring thus:—

In 1891 the 42nd case—of cerebral hæmorrhage.
In 1898 the 95th case—of pneumonia.
In 1900 the 36th case—of pneumonia during incubation.
In 1900 the 37th case—complicated with scarlet fever.
In 1904 the 99th case—of pneumonia.

This increase of severity seems to be entirely due to the concentration of the poison and to the infectious nature of the complications. That the pneumonia of measles is itself infectious, both I and my medical friends in charge of public schools have had ample evidence; and I think this also applies to the suppurative diseases of the ears and other complications. What is true of the hospital ward is still more so of the cottages of the poor, with their very inadequate accommodation; and when, in these, three, four, or more children are attacked at the same time, their chances of escaping what may prove to be fatal complications are considerably reduced. The evidence seems all in favour of letting each epidemic work itself out, and not, by artificial means, to increase the size of those that are to follow.

Scarlet Fever : its Home Treatment and Prevention.

By ROBERT MILNE, M.D.

IN laying before you to-night my plea for the home treatment and prevention of scarlet fever, I shall, firstly, give details of the treatment; secondly, set out the advantages of this treatment; and, lastly, substantiate the treatment.

(A) SOME DETAILS OF TREATMENT.

When first I adopted the method of treatment which I pursue, as lately laid before the medical public, I used carbolic oil 1 in 10. For twenty-five years, however, I have used pure eucalyptus oil. During the first four days in a scarlet-fever case, commencing at the earliest possible moment, I have pure eucalyptus oil gently rubbed in, morning and evening, all over the body, from the crown of the head to the soles of the feet. Afterwards this is repeated once a day until the tenth day of the disease. The tonsils I always swab with 1 in 10 carbolic oil every two hours for the first twenty-four hours, very rarely longer. (In swabbing I place a firm mop of cotton wool, the size of the last joint of the thumb, on the end of a pair of forceps. I thoroughly soak the wool in the carbolic oil, and then swab the tonsils and the pharynx as far up and down as possible.)

(B) THE ADVANTAGES OF THE TREATMENT.

This being the simple and easy mode of treatment, let me say a word or two about its advantages:—

(1) When this treatment is commenced early—and I emphasize the fact that early treatment is vital—secondary infection never occurs and complications are unknown. (How serious a question is that of complications is borne out by a recent remark of a scarlet-fever superintendent. “After a short time,” he said, “we have not only the scarlet-fever-germ infection to contend with, but other germs secondary to this, causing us the very greatest trouble.”)

(2) *Absolute Prevention of Infection.*—With this treatment carefully carried out, I have no hesitation in allowing children to occupy the same

room or even sleep in the same bed ; nor would I hesitate to take milk from an infected farm or dairy, provided the patient had been properly treated.

(3) *The Economy of the Treatment*.—One medical officer of health reported that thirty cases, due to milk infection, cost £300. Another stated that in his town of 70,000 inhabitants such a system would save many thousands of pounds every year. A specialist's view is that it would mean a saving of many millions annually to England alone. The treatment for a case means the cost only of a pint of eucalyptus and an ounce of carbolic oil.

(4) *Its Household Economy*.—The mother is free to attend to both the patient and her duties. The father is free to go to work without the slightest risk, and the children are equally free to attend school.

(5) No after-disinfection is necessary, for, the patient having been disinfected, nothing remains.

(C) SUBSTANTIATION OF THE METHOD OF TREATMENT.

When I left Scotland thirty years ago there was a hazy report throughout the country that if cases of scarlet fever were rubbed with ointment and oil the scales (in peeling) were thereby prevented from flying about, and thus the risk of spreading the infection lessened. Further, that if carbolic oil or other disinfectant were used, this risk was still further lessened. Acting upon this hint, I, along with others, began to use carbolic oil at the very commencement of the disease, and I found, as a result of experience of cases occurring in five different households with several children, that the infection was limited to one member of the family. At first, however, I did not fully realize the value of the method.

As I gave in the *British Medical Journal* of October 31, 1908, a résumé of some of my experiences in private practice, where I showed how safe it was for the children to live and sleep together, I will only here and now give one instance. In 1895, on the tenth day of the disease, I took one of the chaplains of the homes, who was peeling most freely after a severe attack, to my own home on a visit. There he mingled without any restriction for several days with my own children. All of these, I may mention, were very susceptible. None of them then had had an attack of scarlet fever, although four of them had it at different times later. In another private family of seven children, on three different occasions at intervals of many months, three of the children had

scarlet fever without any spread, although they all mingled unrestrictedly with each other.

The remaining portion of my paper deals with the cases of children in Dr. Barnardo's Homes. At present we have over 8,500 under our care. At the Girls' Village Home, Barkingside, Essex, there are at present about 1,300 in residence, the ages ranging from babyhood to 16 years. The village consists of a receiving house and sixty-seven cottages, and each of these cottages is occupied by from fifteen to twenty-five girls, with a lady in charge. The village covers sixty-four acres, and the cottages are widely apart. The various London homes have from 120 to 400 children.

My first connexion with Dr. Barnardo's Homes was due to an outbreak of scarlet fever at the Girls' Village Home early in 1880. I have said that to-day 1,300 girls are there in residence, but then the number was under 500. Among these 500 there were 120 cases. Never since then have we had an epidemic of this proportion; indeed, epidemics are with us of very rare occurrence, and when they do occur the cases are very few. A former President of the Local Government Board asked me some years ago: "Can you explain to us, Dr. Milne, how it is that you have so few epidemics among the children under your care in Dr. Barnardo's Homes? And how, when you do have such cases, they are so few in number? In our schools we have many epidemics, and these affect sometimes one-half, or even three-fourths, of the children in residence." This question drew my attention markedly to the undoubted fact which had prompted it, for it showed very clearly the marked effect of the lines of treatment I was in the habit of following in comparison with the usual treatment pursued by others in similar circumstances.

Since 1880—that is, for twenty-nine years—I have had no such experience with scarlet-fever cases as those I shall now refer to. All have occurred since the appearance of my paper. The record is unique. The cases have been seen and carefully examined, and their histories carefully gone into by some seventeen medical superintendents of scarlet-fever hospitals and by over 200 medical officers of health and general practitioners.

Limitation of Spread of Infection.—Let me remark that the astonishment expressed by these visitors is universal among medical men whenever they have observed cases under the treatment. On many occasions I have kept a child in the same room with from six to twenty-five other children—all under 14 years of age—

without any infection spreading. One case with specially bad tonsils was seen by a medical officer of health, who said, "If tonsils ever convey infection, this would!" Another, after seeing many cases, said, "It is marvellous. To think that I was so long in charge of a scarlet-fever hospital, and in spite of all I could do, and keeping the children seven and eight weeks, to find a fresh outbreak on their return home; yet here, before our eyes, we see case after case in cottages with from sixteen to twenty-five others, mingling freely with one another all the time, and after a few days with thirteen hundred other children, both in school and at church. Our system of treatment seems so ridiculous, with its expense, its labour and worry, in contrast to this simple plan, the efficacy of which is beyond question." Both doctors and public have such a firm conviction of the infectious nature of scarlet fever that I find it strikes everyone with astonishment to discover that, treated thus, it is as little infectious as chilblains. During the year ending October 31, 1909, twenty-four cases occurred in the Village Home. These occurred in eighteen different cottages and in the receiving house. There were three cases in the latter and in one of the cottages; while in both of these, as well as in another cottage, two cases occurred on the same day. Every case had all the ordinary symptoms of scarlet fever, followed by well-marked peeling. None of these cases were isolated. Most of them were treated in their own cottages. The other children visited them daily, while in most instances girls slept in the same room. After ten days the patients were allowed to be out of bed and to mingle freely with any of the thirteen hundred other children in the village at play, at school, and in church. These patients were inspected by many medical officers of health, superintendents of scarlet-fever hospitals, and general practitioners. In the case of one child, aged 4, the symptoms of scarlet fever appeared on the day following her admission to the home. Her temperature rose to 103·8° F., and the case was very severe. On the eleventh day, when the child was peeling most freely, she was seen by a scarlet-fever hospital superintendent and a local secretary of the British Medical Association. Some sixty-nine other children were in the home, sixteen of whom were under 4 years of age, and with these she was at play. The superintendent's remark was "Good gracious, is it safe? I see the scarlet-fever case and I see the healthy children all about in the room, but I must have a few days to think over it, for I cannot take it in. It is so contrary to all my teaching and experience." At the end of the fourth

week she was seen by a medical officer of health with some fifty children at dinner under the trees in the open air. She was inspected two days later by other medical practitioners from a large society.

The final cases occurred in the beginning of October of this year. On October 27 they were inspected, on return from school, by one of London's most celebrated physicians. He remarked, after going round, "How hard it will be for medical men to give up their old practice, although the facts are so clearly set before them! Such an interesting sight I have not seen."

Leopold House Epidemic, 1909.—In Leopold House, one of our London homes, we have 300 boys from 6 to 14 years of age. On February 4, 1909, a case occurred there. The boy was transferred at once by my son to Her Majesty's Hospital in Stepney Causeway, and there the usual treatment was continued. Further cases ensued, until on April 8 thirty-five cases had been recorded. These were treated in two wards, amongst other patients. After ten days nine of these were passed on for isolation to a cottage near, because new cases were still occurring at Leopold House, and because we wanted room in the wards. During the period that these were in our hospital we had over 180 patients in residence. These included fourteen under 5 years of age. The operations performed during that period included the following:—

Removal of large portion of scapula	1
Hernia	3
Cataract needled	1
Osteotomy of femur	2
Tonsils and adenoids removed	1
Nerve-grafting of facial nerve	2
Mastoidectomy	1
Circumcision	3
Excision of knee	4
Movement of elbow-joint	2
Paraffin injection for bridgeless nose	1
Suture of median nerve and separation of tendons	1
Osteomyelitis of upper end of tibia	1
Astragalectomy	1
Fracture of humerus	1
Empyema	1
Subtrochanteric osteotomy of thigh-bone...	1
Bruised torn hand and fingers	1

This epidemic was certainly caused by pen-and-pencil infection, for my son remarked on two occasions that two parallel infections were running. The lads attended six different schools outside, yet all the cases came from two schools. Not one of the lads had a pen or pencil of his own, but got both at school. Within three days after the lads

were kept from these schools there were no more cases. A similar experience occurred in another home. This points to an important but neglected item in school hygiene. I have not found it referred to in any text-book, yet daily in London alone—both in ordinary and higher schools—some millions of pens and pencils are handed out to children. Naturally and always children put these in their mouths, as the most casual inspection will show. Need we wonder that scarlet fever, measles, whooping-cough, mumps, and even diphtheria and syphilis are spread?

Special Incidents.—Special incidents in connexion with this outbreak are worthy of comment. On February 23 a boy was admitted to the hospital to be operated on for hernia. The lad in the next bed on the 26th had been down with scarlet fever for nearly forty-eight hours. When I went round the ward on March 1, I found that the lad had scarlet fever. “How is this?” I inquired. “Has our plan of treatment failed?” It was found that early in the morning of the 26th the hernia patient borrowed the washing flannel and towel from the scarlet-fever case in the next bed, and that morning, before the lad was rubbed with eucalyptus oil, he had used both. The mode of infection thus became apparent. Further, that morning the lad had been out of bed and taken the breakfast to six boys in an adjoining eye ward, where there had been no scarlet-fever cases. He handed them their bread and butter, &c. This occurred on March 1. On March 3 and March 4 three of the lads in this side ward developed scarlet fever. This is the only occasion on which I have known scarlet fever spread after a patient was under treatment, and we need not be surprised at the fact when the circumstances are considered. All the cases had from slight to very heavy peeling. One lad went out without permission, caught cold, and died a week later of double pneumonia. The right lung, we found, was solid; the left lung was nearly so. All the time the urine was normal in quantity, and it contained very slight albumin. The kidneys were in a healthy condition and normal in weight. It is a most noteworthy circumstance that the cause of death was pneumonia—a disease of rare occurrence after scarlet fever—while the kidneys were healthy.

An Otorrhœa Case.—A lad was admitted to the hospital suffering from scarlet fever and chronic otorrhœa. After a few days mastoiditis gradually came on. I operated on Monday, March 22. The mastoid antrum was opened and pus found. The iter was also opened up, but the ossicles were not disturbed. On March 24 he was satisfactory in every way.

An Ichthyosis Case.—The twentieth case at Leopold House was an extreme case of ichthyosis. When the eucalyptus oil was applied it caused such pain that my son wrote particulars of the patient, and sent him to the isolation hospital. This is the only case in which I have found that the treatment was inapplicable.

Medical Inspection.—In connexion with this outbreak I addressed a letter to the secretaries of the Medical Officers of Health Association and of the Medical Officers of Public Schools Association; also to the medical superintendents of scarlet-fever hospitals in London. I explained all the circumstances, gave the list of operations, and conveyed an invitation to them to visit the hospital at 19, Stepney Causeway. In response I had many visitors from the hospitals, who spent hours in investigating the cases. They were amazed to find scarlet-fever patients in beds side by side with these serious operation cases. In some instances the rash was fully out and peeling was progressing in various stages. The history of each case was fully gone into: for instance, the flannel-and-towel case, which was said by several to be most interesting and instructive; also the pneumonia case, which showed how free the kidneys were from attack. Many of my visitors said they were the most convincing evidence of my claims for the benefit of the treatment. The hospital space is 750 cub. ft. per patient. Moreover, we have not through ventilation, while the lads can join hands from bed to bed—a great contrast to 2,000 cub. ft. and through ventilation.

Stepney Home Epidemic.—In the Stepney Home there are some 350 lads, mostly over 15 years of age. Since March 25 to July 5 eleven cases have occurred, while in some of the other homes there have been twelve cases. These were all treated on the same lines, and were mixed with the same class of patients in Her Majesty's Hospital as the above cases from Leopold House. In the Stepney cases the symptoms were well marked, both in the initiatory stages as well as in the peeling. One case in this outbreak calls for special note. The boy came under medical care after he had been ill for some days. His history sheet indicated sore throat for several days with headache, vomiting, and well-marked eruption. Then he was treated in the usual way. On the fifteenth day he had a rigor, pyrexia, and pain at the angle of his jaw. An abscess formed. This was followed by heavy albumin and pericarditis. He died on the thirty-sixth day. To me this does not negative the claim I have made for the value of the treatment. For in this case the treatment was not commenced by any means at the earliest possible moment, and I

always strongly advise the utmost promptitude. It comes into line with many of the hospital cases, for ample time was allowed for secondary infection. May not the question be fairly raised in this connexion: Do not *all* serious complications come through secondary infections? I am inclined to think they do. A medical officer of health saw some of these cases in the various stages and remarked: "Good heavens, Milne, a scarlet-fever case between two cases of recent operations for hernia! I could not have believed it unless I had seen it."

An Experience among Babies.—I cannot resist including the following important testimony: On February 26, 1909, Dr. Charlotte Wheeler, resident medical officer in our "Babies' Castle" Home, Hawkhurst, Kent, reported that they had an outbreak of scarlet fever, and that she was treating the patients on the lines I have indicated. Six cases occurred from February 24 to March 3, and these were nursed together in the babies' room, being sent there as soon as they were found, the nurse attending to the other children at the same time. On March 10 the children all returned to their respective nurseries, and the service-girl began very light work in the nursery. She was peeling freely at the time she started work among the uninfected children. One case peeled heavily, the others more slightly. There has been no nephritis or other trouble except with one child who had been previously suffering from otorrhœa, which had ceased, but recommenced. Although there were some eighty-four children in the home, the oldest being 8 and 9 years of age, no further case occurred. Moreover, they are delicate children, placed there for special nursing.

At Sea.—A very important experience is that of a medical friend who had seen some of the work I have described above. Having charge of an emigrant party to a distant colony, with many children, he found some cases of scarlet fever appear among them soon after starting. He had a good supply of all the necessaries for the method of treatment I have advocated. No case appeared after the third day out. Consequently there was no spread of the epidemic.

Outbreak at our Birkdale Home.—This home is under the care of Dr. F. A. E. Barnardo—a brother of the founder of these homes. The children are all in most delicate or crippled condition, and there were forty-nine in residence. Four of the inmates were attacked in July. In Dr. Barnardo's absence his assistant sent the first two cases to the isolation hospital, but they were brought back on Dr. Barnardo's return and treated. They were placed, with the other two cases from the commencement of the attack, in a room where four other children

slept every night. They were from 8 to under 2 years of age. None of these four had had scarlet fever, yet there was no infection nor complication. I have before me frequent and full reports of urine tested; on every occasion there was no albumin.

It will show the condition of the children in this home when I specify the condition of those children affected. The first suffered from congenital deformity of both hands and feet; the second was very delicate, and had undergone amputation of both legs below the knee; the third weighed $6\frac{1}{4}$ lb. when admitted at 8 months old, and suffered from congenital syphilis; it had also a bridgeless nose, and suffered from ozæna and otorrhœa; the fourth case suffered from Pott's disease of the spine and had a sinus. Those who slept in the same room included two cases of infantile paralysis and two of malnutrition. Their ages were 6, 4, 2, and under 2 years of age. The room in which they were treated was on the same landing as the bedrooms in which all the younger children of the home slept. The nurses went freely in and out among the children all over the home. The youngest and feeblest children, a note says, were chosen for this test. Dr. Barnardo was delighted with the result, and will always adopt the same treatment.

In conclusion, I have given you some details of treatment, the advantages claimed for it, and sufficient proofs, I trust, to substantiate the same.

DISCUSSION.

Dr. GOODHART said he had listened to both papers, which were full of facts, with extreme interest. Of Dr. Armstrong's he could not say much, except that some of his observations bore resemblance to points he had himself noted. He had always said when he went to a public school to see an outbreak of measles that he would rather see anything else, because there were a large number of cases, and he had observed that the danger of cases increased as the epidemic went on. It seemed impossible in the English climate to get the amount of thorough ventilation which was necessary to dilute the poison which was generated. The other facts in Dr. Armstrong's paper seemed of a kind to be thought over and studied rather than debated. But he had seen and talked over with his friend, Dr. Milne, some of his observations with regard to scarlatina, and it seemed to him that Dr. Milne's work at Dr. Barnardo's Homes, provided it were substantiated, was of the most epoch-making that had been done for many years. Personally, he accepted the facts—he did not see how anyone could get away from them—and that being so, it was unquestionably a revolution in the treatment of scarlatina. As he rightly said, if his statements were true, and one could avoid turning the home into an isolation hospital, and putting everything upside down and scattering the

family far and wide, and costing much, it was a comparatively easy way. Dr. Milne rightly said the treatment was not new. Twenty years ago he (Dr. Goodhart) put into his little book on children's diseases a very fair and full description of Dr. Jamieson's treatment on the same lines at Edinburgh.¹ It was not perhaps quite so simple, but Dr. Jamieson, in 1884, made the statement that by inunction of children from the crown of the head to the soles of the feet, and swabbing their throats, he had never seen the spread of any case of scarlet-fever infection during the three years in which he had carried it out. From time to time he (Dr. Goodhart) had seen many cases of scarlet fever, and he had often said, Why not try inunction? The reply had always been that the doctor did not dare. Even after Dr. Milne's paper he believed the practitioner would not dare unless the subject were taken up by medical officers of health. If medical officers agreed with the facts, and that the plan was a successful one, medical officers all over the country should take it up and insist upon it being carried out; then the general practitioner could say he was acting on authority. Dr. Milne said he used carbolic oil for swabbing the throat, 1 in 10 in strength, and that this was done every two hours for twenty-four hours. He (Dr. Goodhart) did not like the idea of that strength being used in a child's throat so often; it could scarcely be free from risk. Dr. Jamieson years ago suggested the use of glycerine and boric acid, and he inclined to think it might be equally good for purposes of disinfection, and have this additional advantage—that in all these throat conditions glycerine often had a marvellously good effect. He regarded the paper as an admirable one, which marked a distinct advance in medicine.

Dr. THEODORE THOMSON, C.M.G., said he was specially interested in the paper of Dr. Armstrong, because in his early days at the Local Government Board he was instructed to inquire into the control of measles in England and Wales. He gathered that Dr. Armstrong was not very hopeful of being able to control measles. But with regard to the observations as to the weight of boys during the incubation period, he believed that, if these observations afforded trustworthy indications, it could be seen by weighing the boys day by day whether they were going to develop the disease, and that the outbreak could be checked by timely isolation of those presenting the variations in weight remarked by Dr. Armstrong. He would have liked Dr. Armstrong to supply a few more facts: how many boys he had under his observation, how many were weighed daily, what proportion of them lost weight during the incubation period, how many of them were attacked, and what happened to the remainder?

Dr. PARSONS said that one point in Dr. Armstrong's paper which seemed open to a difference of opinion was his remark as to the uselessness of trying to prevent an epidemic of measles, because if they were to have the disease they might as well get it over quickly. It was owing to the prevalence of that feeling that measles still remained with us and caused such a high mortality. By far the larger mortality from measles occurred under five years of age, and

¹ Goodhart, J. F., "Diseases of Children," 4th ed., Lond., 1891, p. 181.

mostly in the second or third year of life. If the period at which the child had the disease could be postponed to a period when it was found to be less fatal, the general death-rate from it must diminish. If medical men could succeed in checking epidemics in public elementary schools, more children would be saved and the fatality from the disease would be diminished. It had been stated that epidemics in schools in London were not found to occur unless the proportion of unprotected children reached 30 or 40 per cent. When that proportion was reached if measles was introduced, an epidemic occurred, until the number of unprotected children in the school was reduced to between 15 and 20 per cent., and then it declined.

Medical officers of residential institutions had the advantage that they could watch cases of measles and scarlet fever from the first, and get hold of them in the earliest stages, as compared with the medical officer of health who could only look after them after receiving notification. Dr. Williamson had shown that of the secondary cases of scarlet fever occurring in households, by far the greater number were contracted before the removal of the first case. In that way the failure of hospital isolation to check the spread of scarlet fever was explained. With regard to Dr. Milne's paper, the eucalyptus-oil treatment of scarlet fever was by no means new. A paper was read before the Epidemiological Society in 1889 by Mr. Brendon Curgenvin, and he claimed that by it isolation and disinfection would be unnecessary in the case of scarlet fever. There was another paper, by Dr. Joseph Priestley, in 1895, who, while not endorsing all that Mr. Curgenvin had claimed for it, still considered it useful; yet it had not come into general use. If isolation had not been rendered useless, was it because the treatment had not been carried out in the right way, or because the cases were not secured sufficiently early to prevent infection occurring? He did not know how far the throat treatment explained the better results claimed by Dr. Milne. At present more attention was paid to throat infection than to skin infection. Infection by means of pencils was well known to school medical officers; and mention of it was to be found in the official memorandum of the Local Government Board on schools in relation to infectious diseases.

Dr. F. M. TURNER said that many persons had accepted Dr. Milne's invitation to inspect his cases. If any of them had had the courage to follow Dr. Milne's lead, he hoped they would give the Section the benefit of their experience. He, personally, had been so impressed by Dr. Milne's results that he thought the treatment should be tried in a scarlet-fever hospital, and he began his experiment in March this year. Dr. Milne's contention was that his treatment could be used instead of isolation. This contention, of course, could not be tested by experience in an isolation hospital on ordinary cases; but there were three aspects of fever-hospital work to which Dr. Milne's theories, if correct, ought to apply. Firstly, the fever hospitals receive a large number of doubtful cases, and mostly treat them in single-bed isolation rooms. If eucalyptus was a preventative of infection, such cases could with impunity be treated in the general fever wards. Secondly, all fever hospitals find that a

certain proportion of their discharged cases, usually from 3 to 4 per cent., are followed by fresh outbreaks of scarlet fever in the home. If Dr. Milne's theory were correct, they would be able to prevent entirely these return cases. Thirdly, small outbreaks of scarlet fever frequently occurred among the children in the diphtheria wards. To these Dr. Milne's treatment, instead of isolation, was strictly applicable. Since March he had had under his own care 152 cases admitted, which he rubbed with eucalyptus oil. Of those, seventeen were still under treatment, and 135 had been discharged or had died. Two died and 118 were sent home, and fifteen were sent to a convalescent hospital. At his request, one of his assistants started the treatment in an adjoining ward; and he had now had fifty completed cases. So that the total number of completed cases was over 180. As regards the mixing of doubtful and certain cases, in his own ward he had treated eighty-nine certain cases of scarlet fever, seventeen doubtful cases, and twenty-nine who were at first doubtful but finally considered not to have scarlet fever at all. Only one of these caught scarlet fever. Dr. Lakin, however, had not been so fortunate. Among his fifty cases only seven proved not to have the fever, and of these seven, two subsequently developed it. As regards return cases, he had discharged four, at the homes of whom an outbreak subsequently occurred. One was a girl of 14, who had a definite rash and peeled slightly, and by the eighteenth day the peeling was practically complete. Another was a girl of 10, a moderately severe case, but with very free peeling, and twenty days after the onset she was well. He wrote to the medical practitioner who sent her in, saying he had treated her by the new method, and suggesting that she should be sent home. He replied that he would much rather he (Dr. Turner) kept the case a little longer. She was therefore kept ten days longer. She was discharged on the thirty-fourth day, and not very long afterwards another case occurred in her home. Another patient, a boy of one year, was oiled in the correct manner, and was discharged on the sixteenth day; there had been no peeling, and Dr. Turner saw no rash. The mother took him home, and later the doctor who certified the case telephoned that he saw typical peeling. He saw the child the same afternoon, and on two occasions tried to see the peeling, but failed. Six days later his sister developed the fever. The fourth case was that of a boy aged 4, who was discharged on the fifteenth day of the disease, and he had had much trouble and correspondence about that. They gave, on his own discharges, an infectivity rate of 3·4 per cent. Including Dr. Lakin's fifty cases, the rate came down to 2·9 per cent.—not very different from what they were used to. They might have been expected to be a little under the average, because the worst return cases occurred in the winter, and his testing of the cases ran from March to the present time. After the treatment had been in use two or three months he had, within a week, three notifications of return cases. His discharges had been forty to fifty, showing a 10 per cent. rate: that was so much more than they were used to that he feared he might lay himself open to a charge of malpraxis if he went further. The cases treated by Dr. Milne's method were discharged earlier than usual—from fourteen days up to thirty-five days or more—not after ten days, as

Dr. Milne considered safe. As regards the prevention of return cases, the experiment was a partial, if not a total, failure.

As regards scarlet fever in the diphtheria wards, he had used the treatment several times on doubtful cases; but the first certain case that he had treated thus and left in the ward with other children—twelve of whom were under 5 years of age and five over—was a little girl, admitted on July 14. Two cases of scarlet fever had broken out in that ward on August 25. These two were removed to a small isolation room, and they, with all the other children remaining in the ward, were oiled for ten days. On September 2, six days after oiling commenced, Ivy Morgan developed a severe attack of scarlet fever. She was not removed, but treated throughout with the other children. She developed nephritis, which caused her to be detained for a long period, and she was discharged on November 18. No cases of scarlet fever occurred until November 8, two months after Morgan developed her disease. This patient was oiled instead of being isolated, but the oiling was a distinct failure, as a further case developed on the ninth day, two cases on the tenth day, and another—the fourth—on the eleventh day. In all three respects, therefore, in which his experience had been used to test Dr. Milne's treatment, it had been at least a partial failure. As regards complications, he did not find any great difference between the cases treated and untreated. In his own ward he got a higher than average rate for albuminuria, abscess of the neck, and tonsillitis; a lower for otitis, adenitis, and nephritis. Dr. Lakin found an increased rate in three complications out of four. He did not attach much importance to those complication-rates, because the children attacked were under the average age. He was more concerned with the discrepancy between Dr. Milne's facts and his own as regards infection. He mentioned several possible causes of the difference, but did not regard any of these as a satisfactory explanation. He hoped that further work might be done until the question was cleared up. He congratulated Dr. Milne upon his courage in bringing the matter forward.

Dr. BIERNACKI stated that the method was being tested at Plaistow Hospital. Inunction was employed in the case of every patient admitted to the hospital, whether the case was certified as suffering from scarlet fever or from some other disease. This was to safeguard patients in the wards reserved for diseases other than scarlet fever from contracting that disease in event of an unrecognized case of scarlet fever gaining admission to these wards. Nevertheless, patients were infected by scarlet fever in them. Again, in the scarlet-fever wards 700 cases were dealt with by inunction. There were many doubtful cases in these particular wards, and five of them acquired scarlet fever from the undoubted cases. Further, on sending undoubted and doubtful cases to the convalescent home, five of the latter developed scarlet fever there. Five members of the nursing staff who were carrying out the treatment developed scarlet fever. While the treatment was going on there was an increased prevalence of septic complications—particularly otitis—among the scarlet-fever patients. The number of return cases also rose above the average.

Dr. BUCHAN said the paper of Dr. Milne was not so important to medical officers of health as regarded treatment as from the point of view of the facts which he (Dr. Milne) set before the Section. Those facts showed that scarlet fever was a disease which was much more infectious during the first ten days than it was during the later periods. Recently he had a case of scarlet fever which was kept at home for a fortnight, and attended school for the three succeeding weeks, with no effect in that school. He did not suggest that one should therefore allow all cases of scarlet fever to go after a fortnight's isolation broadcast amongst the population, but he mentioned it to show how relatively slightly infectious scarlet-fever cases were in the peeling stage, provided there were no discharges from the mucous membranes. With regard to the point made by the author that the mother should be free to attend her household duties and the father to go to work, he did not see that the treatment made any difference to that. Statement No. 5 had nothing to do with the treatment which Dr. Milne pursued, but it was important from the point of view of public health. He would like Dr. Milne to say what the rationale of his treatment was. With eucalyptus and carbolic oil in use there might be an idea that disinfection was intended. But neither carbolic oil nor eucalyptus oil was a true disinfectant. He did not see what evidence there was that a patient was infected by means of a washing-flannel when he understood he lay in the next bed to a patient who had scarlet fever.

Dr. MEREDITH RICHARDS remarked that Oliver Wendell Holmes had said that homœopathy had one advantage—namely, that it gave one the opportunity of becoming acquainted with untreated disease. He believed this largely applied to Dr. Milne's methods. He (Dr. Richards) did not think anyone could explain how inunction could have any benefit in scarlet fever. It was a case in which one ought to assume that the cogency of the argument and the amount of facts produced should be in proportion to the departure from the probabilities; and he thought Dr. Milne had not brought forward sufficient data. If one was to believe that the inunction of eucalyptus was effective in bringing to an end an infection by scarlet fever, the disease must be assumed to lie in the skin. Yet he thought it was generally agreed that the skin was not infectious. That was proved by the fact that desquamating children frequently attended schools and lived with unprotected children without infection taking place. And it was known that infection in scarlet fever was present before desquamation began. Desquamation, he believed, was due to a chemical toxin. He had failed to become convinced that swabbing of the throat could be efficacious to the extent that they were asked to believe. Diphtheria throats could be swabbed by the week or fortnight, and experience seemed to show that swabbing had little or no effect on the bacilli. The bacilli were often in crypts $\frac{1}{2}$ in. below the surface of the tonsils, where swabbing could not penetrate. He knew of no reason why they should be more successful with scarlatinal throats.

Dr. CROOKSHANK said he agreed with what Dr. Meredith Richards had said: that members had had an opportunity of seeing what untreated disease was like. He thought Dr. Milne had certainly proved that when once the horse

had been stolen it mattered very little whether one locked the stable door or not. Once the outbreak had started, Dr. Milne had not perhaps got worse results by treating scarlet fever with eucalyptus oil than other people had obtained with other methods. The title of the paper was "Scarlet Fever: Its Home Treatment and Prevention," and he understood that Dr. Milne's conclusion, from his observations, was that scarlet fever could be safely treated at home, and even that father could go to work and mother could attend to her household duties. But he did not think Dr. Milne had brought forward a single point to show that such home treatment could be safely conducted. The results given were from treatment at hospitals with skilled nursing. He asked what steps had been taken in the wards to prevent infection by sprays, spoons, glasses, &c. That 30 out of a community of 300 fell within a month or two was very much the usual experience. When scarlet-fever cases occurred in diphtheria wards, granting there were proper nursing, skill, and observation, infection did not take place nearly so frequently as the general public thought. Dr. Milne's results should be compared with experience in isolation hospitals. The superintendents of such institutions do not admit that scarlet fever spreads with extraordinary rapidity when the patients are kept in bed, with the observance of certain simple rules.

Dr. J. C. THRESH wrote with regard to Dr. Milne's paper that the treatment of scarlet-fever patients by inunction with eucalyptus oil had interested him for several years, and when he had charge of the small isolation hospital belonging to the Chelmsford Rural District, he used it in nearly all cases, and, so far as he was aware, there were no return cases. During one epidemic the hospital was overcrowded, and about a dozen patients were accommodated in a tent. All the patients did extremely well. He formed a very pronounced opinion upon its value, but whether it rendered a child non-infectious after ten or twelve days he was not able to determine. The popular opinion that a child is infectious so long as it shows the slightest sign of "peeling" is so strong that he did not dare to send out a child until desquamation had practically ceased. Many medical officers in hospitals would, he believed, try the experiment if they could feel certain that they were not laying themselves open to an action for damages if a return case occurred. The enormous pecuniary and other advantages which would accrue to the community if Dr. Milne's claims were substantiated, as he believed they could be, led him to hope that some means might be devised whereby the treatment could be tested under conditions which would render the results conclusive.

Mr. ARMSTRONG, in reply, said that in an examination of a series of suspects by the weight test, a marked difference had been observed between those who were developing measles and those who, for the time, escaped. Referring to the remarks of Dr. Parsons and the President, as to the great mortality from measles in infants up to two years of age, his contention was that it was due to pneumonia and similar complications, produced by the concentration of the poison from the aggregation of a number of cases with inadequate accommodation.

Dr. MILNE, in reply, said that one or two comments had been made as to the alleged risk of using 1 in 10 carbolic oil both in regard to the throat and to the kidneys. He had used it for thirty years and had never found that it gave pain or caused trouble in any way. In fact, it greatly relieved pain and enabled the patient to swallow with comfort. Drs. Turner and Biernacki had given reports of their experience in isolation hospitals. One important point to be borne in mind in this connexion, however, was that in hospital practice the medical attendant is unable to apply the treatment at the earliest period of the disease, and he regarded that as of vital moment. Consequently, such cases as had been reported upon came under the category of secondary infection, regarding which he had asked in his paper: "Are not *all* these complications caused by secondary infection?" The dangers of pen-and-pencil infection, it was said, had already been recognized. But what had been done in the matter? We could not insist too strongly or urgently upon the necessity for absolutely doing away with this real and horrible danger. By it millions of children are daily exposed to numerous deadly diseases. Since his experience of two epidemics undoubtedly arising from this source, every child in the schools under his medical care had its name on both pen and pencil. There was no transfer of one to another, and consequently all risk of infection from this source had been eliminated. He had never urged that his proposed plan of treatment should be taken up indiscriminately. He had merely set forth his experience and had repeatedly asked that it should be tested under the united care of the medical attendant and the medical officer of health. Dr. Curgenven's method had been mentioned as if he used pure eucalyptus oil. What he used was Olenshaban oil, but he found it fail (as Dr. Priestley did) and had to return to what he had used for years to re-test thoroughly his former conclusions. The question was asked, "How was it that in his former paper the ages of the girls at the Girls' Village Home were given differently from those he had given to-day?" As he had mentioned in this paper, many babies were constantly passing through the receiving houses, where the case upon which he commented occurred. The child, therefore, was constantly in contact with sixteen other children of under four years of age.

Epidemiological Section.

January 28, 1910.

Dr. JAMES NIVEN, President of the Section, in the Chair.

The Control of Scarlet Fever.

By F. G. CROOKSHANK, M.D.

I AM indebted to you for the opportunity of stating opinions which are unlikely to command your immediate assent, and only regret the unfortunate circumstances which have led to the hastened preparation of my notes at a time when official and non-official tasks are more than usually onerous.

Occurrences, such as are within the experience of most of us, have led to the recognition of definite drawbacks to the attempted control of scarlet fever by removal to isolation hospitals. Statistical inquiries have led some to question altogether the usefulness of such removal; and, quite recently, we have in this place listened to a paper in which the author sought to establish the *needlessness* of any attempts being made to control the disease by removal. We have also to recognize a more or less coherent mass of lay opinion which is inclined to question the value of isolation hospitals, so far as scarlet fever is concerned, for the somewhat inconsequent reason that such hospitals are rather expensive institutions; and laymen who adopt this attitude are not prone to minimize the force of professional criticism of isolation-hospital methods generally, and the hospital method of scarlet-fever control in particular.

It is necessary therefore that we should clear our minds of that professional cant which is as odious as cant in any other form.

My own opinion is that, even should it be proved that the removal of cases of scarlet fever to isolation hospitals does not generally lessen the incidence of the disease in the district served, yet still the provision of such hospitals, even by the most extravagant of municipalities, is about the cheapest thing in practical insurance offered to the small ratepayer; since, for about five or six shillings a year, it insures him against all expense in the event of his household becoming stricken with infectious disease, and safeguards him against business or professional loss.

But I do believe that the removal of cases of scarlet fever, if properly carried out, does reduce the incidence of the disease in the community served. I know it has reduced the case mortality, and I think there is fair evidence that it has modified the type. I am convinced, moreover, that many, if not all, of the accidents or incidents of fever-hospital segregation are as absolutely preventable as are the septic catastrophes which do not now occur in general hospitals.

I do not propose to-night to attempt the substantiation of all these beliefs; but am bound, as we all are, to admit that it is our duty, when attempting the control of disease in the interests of the community, to see that we achieve the irreducible minimum of the drawbacks that in all cases attend human endeavour, and in this case fall very hardly on individuals. It is clear enough, too, that if we can at the same time save expenditure, we shall gain a more ready ear when we ask for power to administer.

The control of scarlet fever in a community, so far as our present knowledge allows us to proceed, means the control of seasonal prevalence; the limitation of occasional outbreaks, and the destruction of endemic or sporadic foci. It involves special procedures: the organization of a public-health department and the particular adjustment of its relations to the practitioner; the establishment of an isolation hospital and the co-ordination of its administration with that of the public-health department; the administration of the isolation hospital so that complete control is obtained over the diseases admitted thereto.

Before discussing the way in which scarlet fever may be controlled *by* isolation hospitals and *in* isolation hospitals, it is necessary to state some epidemiological considerations.

We cannot learn the natural history of scarlet fever in either the hospital or the laboratory. We can in these places learn facts which, set in due relation to others, are of importance. But the bacteriologist, or the hospital superintendent pure and simple, is no greater authority

on the natural history of the disease than is a keeper at the Zoo on the habits of the tiger in his jungle. In fact, unless we are prepared to admit that our well-meant efforts are all beside the mark, we must acknowledge that modification of the natural history of disease follows interference by the public-health service. Plague and diphtheria, for instance, are two diseases of which the epidemiology has been profoundly modified by human agency, intentionally and unintentionally, within the memory of the present generation.

The effects of hospital treatment of scarlet fever as carried out during the last thirty years or so are probably less obvious than would have been the case had domestic isolation not been practised for years previously in some degree; and for almost the last vivid account of scarlet fever in a natural state in the United Kingdom we have to turn to the writings of Graves. Here we learn that in Ireland, in the early part of last century, scarlet fever was observed to occur in epidemics; appearing in the late autumn, and dying away in the next spring, after a course of about seven to eight months; reappearing in successive years with raging intensity, and then perhaps disappearing from a district in epidemic form for years together; and in sporadic cases, often of marked severity.

It is worth noting that at this time all grades of severity were noticed, so much so that Graves formulated the axiom that "a constitutional affection, produced by morbid animal or vegetable poisons, may display its evidence by only one or two of the numerous symptoms which usually accompany it." And it is interesting to remember that the cases of this character appear to have been noted chiefly in connexion with the epidemics.

If we now turn to the tables furnished in the last edition of Sir Clifford Allbutt's "System of Medicine," it is seen that the curve of seasonal prevalence of scarlet fever still extends in London, as it did in Ireland a hundred years ago, over a period of seven or eight months. But in Ireland the months of prevalence were usually November or December to May or June. In London the months are June or July to January or February. Almost invariably, in fact, scarlet-fever outbreaks of any magnitude (other than those due to milk) run for about eight months from first to last, and the period of greatest activity is always about six weeks.

Now the ideas with which, as students, we became imbued were, firstly, that scarlet fever always breeds true; secondly, that a case of scarlet fever of the present day is in direct descent from the first case of

scarlet fever that ever existed; thirdly, that outbreaks or epidemics are due to neglect of sanitary precautions; and, fourthly, that epidemics come to an end after undergoing a process known as stamping out. Lately another belief has become current, of which the profession usually seems to endow its holder with extraordinary self-satisfaction; it is that epidemics come to an end from exhaustion of the soil, or, as others say, the number of susceptible persons exposed to infection. Of course much virtue rests in the use of the word "susceptible." Any person exposed to infection at a certain time, and not obviously infected, may be said to be, or have been at that time, "not susceptible." The "soil" varies in different persons, and in the same persons at different times, no doubt; and "exposure to infection" is by no means a fixed quantity, and cannot easily be defined. But does not the infective potency of the virus itself vary; or, what perhaps is not quite the same thing, does not the power of the virus to produce certain "specific" effects vary?

The teaching of Pasteur's classical experiments with rabies and chicken cholera is very clear; and, as Professor Ritchie has well put it, in the laboratory an organism obtained from a lesion of malignant type soon loses its virulence, or is less virulent than an organism from a less severe case of the same disease. Attenuation results from continuous growth on the same media; but virulence may be increased by passage through a series of bodies, or by variation of the nutrient media. Moreover, while the virulence may be increased by such passage, continuous passage may lead to permanent attenuation.

I would ask all those of clinical experience if they have ever known the virus from a typical case of scarlet fever, after passage through two removes, to reproduce in a fourth case the typical symptoms of scarlet fever? With an exception to be presently mentioned, I never have; and I am not sure that, outside the limits of this exception, I have ever seen a *chain* of three typical cases of the disease. The exception is in the case of isolation hospitals. A first return case may apparently give rise to a second return case, and I have known of third, and heard of fourth return cases, making series of four or five cases in all. But these exceptions really do seem to prove the rule; for the isolation-hospital experiment is not a pure one.

Assuming that the series of return cases are not capable of other explanation, and that the third or fourth return cases are not delayed secondary ones, there is always the possibility, if not the probability, of a return case being, not a return of the original virus, but of another

strain of the same virus, carried by an imperfectly immunized patient. It is true that families in which return cases occur are often families in which there is marked susceptibility to infectious disease; and one cannot refuse to admit that a virus, attenuated to a certain degree, may yet, on certain soils, produce a response otherwise not obtained. The hospital occurrence is, however, in fact directly analogous to the laboratory experiments, in which, when making subcultures, accidental contamination and reinforcement occur. And, until or unless I can hear of an authenticated chain, I am prepared to affirm that the virus derived from a typical, developed case of scarlet fever, after passing through two, or at most three, removes in the human body, loses, if not its infective potency, at any rate its power of reproducing typical scarlet fever on normal soil. The virus, in fact, becomes attenuated, or recessive.

There is another clinical observation which many family practitioners will confirm. It is that a process of intensification often occurs. This Graves clearly saw without understanding.

One child in a family has a sore throat; there is no rash, no peeling, little fever. Another child, a week or two later, has a sore throat, greater fever, and a peeling tongue. A third child develops scarlet fever, as we know and describe it. Surely this sequence is but the intensification of a virus in the bacteriological sense.

Is it not within the experience of every general practitioner that such a series frequently occurs at the beginning of an "outbreak" of scarlet fever? Is not the observation repeated year after year in different schools and communities? Is it not a fair deduction that epidemics of scarlet fever (again, I am not speaking of milk outbreaks) follow the intensification of a virus by passage through the bodies of susceptible or, if you will, responsive persons, and that such epidemics terminate by reason of the attenuation of the virus after passage through a further series of bodies? I think there can be no doubt of it, and that the series, from a first manifestation of the virus to its disappearance, usually consists of about six cases, of which the third and fourth are commonly the most "typical" and the most severe.

Put in another way, the normal cycle of epidemic scarlet fever, or so much of it as we can at present recognize in human beings, does not extend beyond a sequence of six cases. The earliest and latest are the mildest and least typical; but infective potency lessens with attenuation. Through what phases the virus may pass, or in what media it may exist after it is lost sight of, is matter for examination. It may perish;

it may continue its generation in human beings with other or no clinical manifestations until intensification occurs in response to variations of the soil or other conditions; it may have an external existence, in the humus or elsewhere; it may continue through other living hosts. Observations do seem to afford evidence that the virus does, or may, pass through a phase in cattle, but whether this be a necessary part of the full cycle or only an accidental side-tracking is, for the present, pure speculation. Neither can we say whence the virus of the first intensive case comes, nor what is the relation of marked sporadic cases to the epidemic cases or those of seasonal recurrence.

I am bound to say that, if one be asked to correlate scarlet fever with any one hygienic, or rather unhygienic, factor—in the way that relapsing fever is correlated with famine, typhus with foul air, typhoid with excremental pollution, cholera with water, and plague with the soil—one would say that there does seem to be a relation between sporadic scarlet fever and dust. Certainly cases of sporadic scarlet fever have been, in my experience, seen far more frequently in dusty houses of a peculiar type than in any others.

If it be true that in outbreaks of scarlet fever, and during the ordinary seasonal prevalence, there is first an intensification and later an attenuation of the virus, certain facts are at once explained. If the usual sequence be a series of six cases, the probable maximum duration in time of such a sequence would be about thirty-two weeks, and the probable shortest duration of such a sequence about six weeks. The mathematical calculation can easily be made, assuming that the period of incubation varies from two to four days in cases of direct contact, and that infective potency is usually present on the second or third day, and rarely after the sixth week in pure infections; and I think it will then be found that an outbreak ought to extend over about the eight months usually observed, and that the maximum number of cases and the greatest increase in the number of cases ought to be observed within a limit of five or six weeks. Such, indeed, is the case every year in London, and everywhere else, so far as I know. It will be found, too, I believe, on inquiry, that the termination of outbreaks from attenuation of the virus, and consequent loss of infective potency, is a far more reasonable explanation than is the alleged termination from exhaustion of the number of susceptible persons. In fact, the latter hypothesis can only be sustained by question-begging about what "susceptibility" really means, and it is certain, I think, that in school outbreaks the proportion of persons exposed to infection in the second

stage of the outbreak who, unprotected by previous attacks, yet escape, is too high to be explained away by talk of "natural immunity."

We have, too, a simple explanation of the fact that many private practitioners and laymen have a strong opinion as to the infective potency of scarlet fever. They are those who see and recognize the intensive cases, and know, too, that the cases secondary to them are more virulent than the primary ones. On the other hand, specialist hospital superintendents, who, for obvious reasons, see more recessive cases than intensive ones, do not share what they consider the exaggerated fears of the layman and practitioner, and pass from scarlet-fever wards to those devoted to diphtheria or other diseases freely and with impunity. But it is the atypical, intensifying case which finds its way through mistaken diagnosis now and again to the diphtheria wards, and sets up the crop of "cross infections" which we all so justly dread.

I think, also, that I can see the explanation of the very interesting observations so recently laid before us by Dr. Milne. I do not wish to minimize the value of antiseptic applications to the throat, and shall, indeed, presently refer to the subject as of cardinal importance. But is it not clear that the cases treated by Dr. Milne with such apparent success are really recessive cases, and not intensive ones? The crucial test of the value of Dr. Milne's method would be, it seems to me, not the treatment in general wards of marked cases recognized *after* outbreaks are well established, but the treatment by his method of mild, atypical, intensive cases. The application of the method should, in fact, *prevent* outbreaks in the community affected, and not merely allow demonstration of the relative impunity with which developed cases can be treated in general wards when once the virus is undergoing attenuation. Indeed, Dr. Milne's results, and the results claimed for public-health administration without isolation hospitals, seem to me to have one and the same explanation. There is no doubt at all that, when once an outbreak has started, and typical cases are recognized on every hand, the outbreak will terminate with mathematical certainty, and that remarkably few so-called secondary cases will occur, whether the cases be treated in a general hospital with inunction or at home without it.

But the magnitude of the outbreaks, seasonal or accidental, should be compared with those in districts where the methods I propose to discuss are in vogue, and the question may well be asked: Is it certain that in cases treated at home without apparent mishap, the virus may

not be transmitted in forms and manners not perhaps obvious, but yet dangerous; and does not strict aseptic hospital treatment offer the greater probability of destroying the virus—of making what a fireman calls a “good stop”? In my own district I know that when, in the autumn, every notified case, however mild, is admitted to hospital, we shall get through the winter well; and that our seasonal prevalence is greater when we commence with a few mild cases which are kept at home by practitioners who assume an attitude of scepticism, which they think advanced. The connexion between the various cases may not be demonstrable, but the correlation of the facts is undoubted, and the conditions of the district for which I am responsible are so far peculiar that I have reason for confidence in the observation.

It is possible, and indeed probable, that the phenomena of intensification and attenuation may be, in the case of diseases other than scarlet fever, manifest to those who have eyes to see and ears to hear. There are many observations, especially in the older text-books, that are deserving of collation, but I allude to them now only so far as they incidentally support the notions I have endeavoured to express.

Before attempting to apply the consideration of these ideas to the practical control of scarlet fever, there are some clinical features to be taken into account, which are of importance chiefly from the point of view of hospital administration. I am accustomed to recognize cases of scarlet fever with tonsillar exudation, ulceration, pellicle, or membrane, as impure cases of the disease. In other words, I believe the pure scarlatinal infection gives no obvious exudation. Some cases without exudation may be, and probably are, impure infections: but this by the way. Cases of pure scarlatinal infection may vary in severity from the mildest degree to the most fatal, and no one will dispute that both the mildest and the most toxic case may run their course without any obvious exudation. Cases with exudation are to be recognized as dual, or even multiple, infections—symbioses if you will—and may be spoken of clinically as diphtherial, diphtheritic, rheumatic, septic, ulcerative, follicular, herpetic, or erysipelatous. Bacteriologically, the “plus” infection may be recognized as due to the Klebs-Löffler, the Hoffman, or the Vincent organisms; to pneumococci, to staphylococci, or to streptococci of various kinds and strains.

We have, however, to recognize, and especially in hospital, not merely the initial coincidence of two or more infections, but relapses, second attacks and minor phenomena, due to secondary infections with scarlatinal virus of perhaps another strain; and in addition the late occurrence

of heterologous infections. What influence these infections, homologous or otherwise, have on the original virus is hard to say. They may, of course, modify it profoundly, and so interfere with what I have called the natural cycle of scarlet fever.

I do not attach the slightest importance to peeling itself as infectious, but see no reason why flakes of epithelium should not be an excellent vehicle for the transmission of organisms derived from the mouth, nose, or ear, and to that extent I believe that skin peeling may be "infectious." So far, perhaps, the injunction of the body may be justified; but I hold it better to attack the mouth and nose directly rather than to dissipate attention to the skin; and the keynote of success in dealing with scarlet fever as it affects the individual is to regard it as an infection of the mouth (or nose) through a break of the surface, except in those rare but undoubted cases where it is found as an infection of a wound of the cutaneous or uterine epithelium. It cannot be doubted, of course, that dissemination of the specific virus may be aerial to the extent of the radius of expired droplets, or that substances and fabrics on which droplets, &c., fall can, in accordance with their suitability as a culture medium, convey infection across distances and through lengths of time. These facts are surely as obvious as are the commonplaces of the operating theatre; but it is, also, one of these commonplaces that an article sterile in itself may transfer a virus from wound to wound with disastrous results.

Attempting now to deduce principles for the control of scarlet fever so far as our present epidemiological and pathological knowledge allows, one must commence with the proposition that the existence of the well-managed isolation hospital is justified as the best means of destroying the virus known to us, short of projecting the patients into space for eternity.

It is quite clear that administrative control can only affect to a very limited extent the patient retained at home. The immediate removal of the patient allows for the prompt action of the public-health department by way of destroying any traces of virus in the house. But the prompt diagnosis and notification of the disease, and the examination and, so far as can be, the disinfection of persons in the house, depend on the relations between the practitioner and the health department. It is obvious that the main idea to be impressed on the practitioner is that cases are not necessarily infectious in proportion to their severity, and that the early, atypical, intensifying cases are probably those in which the virus has the highest infective potency, although the amount of

virus (but not the degree of potency) may be greater in the later cases of an outbreak. Every effort therefore has to be made to encourage early and confident diagnosis, and the immediate transference of "first" cases to hospital, however mild or irregular they may seem to be. It is not necessary to labour the importance of each half-hour that passes between diagnosis and removal; the proof is the frequency with which secondary cases are notified in the first week. Nor should it be necessary to labour, in the interests of the hospital itself, the absolute necessity for the ambulance and its arrangements being ordered with the same degree of aseptic scrupulosity that obtains in the operating theatre of a modern hospital. Perhaps it is best not to inquire how often the ambulance of an isolation hospital is truly an aseptic one. Still, though secondary infections due to hospitalism begin in the ambulance and with the ambulance nurse, it is at the hospital gates that the most obvious practical difficulties begin.

In the general hospital the only necessary basis of classification is that of sex, but in the isolation hospital we have had of late years to start with the number of prime infections we propose to treat; to double that number to give us wards for each sex and each disease; to add again for division into convalescent and acute classes; and to make yet further additions for the reception of mixed or doubtful cases, before we can arrive at the number of ward-blocks and wards necessary in the case of the particular district that has to be served. When dealing with hospitals of large size, projected to serve large districts, the proper provision of an adequate number of isolated pavilions does not give rise to great difficulty other than that of expense in building and administration, for hitherto the idea has, of course, been to provide a separate ward for each disease or combination of diseases likely to be treated, and so theoretically to provide for a number of maxima, although experience shows us that not all these maxima are likely to occur together. But, in dealing with the needs of a district of, say, 40,000 or 50,000 inhabitants, the certainty that one will be lucky if allowed one bed per thousand of the population makes the allotting and planning a task of the greatest difficulty. Naturally enough, the apparently logical solution has been to provide a separate ward for each patient, or, in other words, a series of cubicles. This plan has, for small districts, the great advantage that it allows economy in the numbers of the staff maintained, for the strict application of the old plan of "one disease, one ward-block, one staff," results not infrequently in the provision of more nurses than there are patients.

The objections to the cubicle system seem to me to be two: (1) material—that of expense in construction; (2) moral—that the attention of the nurses becomes fixed on the partition as the safeguard. The barrier system adopted by Dr. Biernacki at Plaistow is free to a great extent from these objections; but the question I desire to raise is whether we may not in small hospitals, at any rate, if we get a firm grip of the real principles, safely treat all diseases side by side. I am convinced that we may. If this can be done generally, instead of having to provide for a series of maxima, only one maximum needs to be provided for—the greatest total number of cases likely to need treatment at one time. This would mean probably not so often one bed per 1,000 as one bed per 1,500 of the population.

I recognize that the complete trial of the plan I suggest would mean the planning of a new hospital, and I have had, of course, at Mortlake to make use of pre-existing buildings—separate pavilions separated by considerable open spaces, and providing thirty-six beds on four acres of site. Having these several ward-blocks at my disposal, I have not as yet deliberately placed cases of scarlet fever and diphtheria side by side; but I have treated cases of diphtheria, typhoid and erysipelas or puerperal fever in one ward, and, on the admission of doubtful cases of scarlet fever or diphtheria into the wards nominally allotted to these diseases, I have not transferred them when the diagnosis has been found erroneous. Such transfers may do more harm than good, for obvious reasons. But we have done away with any distinction between diphtheria and scarlet-fever nurses, and therefore to that extent no longer separate *wards* or *diseases*. We isolate *patients* without any mechanical apparatus such as glass walls, or barriers, but by ritual observance only. It is not that one regards the transference of prime infections as a light matter; the idea is that it is as grave an affair professionally for a simple case of scarlet fever to acquire a pus infection as for a case of diphtheria to acquire typhoid or scarlet fever. Each patient therefore is isolated in his or her *bed* without partition or barrier. This can be done, if proper technique is observed, as easily as it is done in the surgical wards of a general hospital every day. Nurses can then pass from block to block, ward to ward, and bed to bed as simply and harmlessly as does a surgeon from a case of empyema to a laparotomy. They relieve each other as occasion may require, give assistance to each other, and interchange quite freely. The saving in staff expense alone is very considerable, and the general results are perfectly satisfactory.

The hospital of which I have control is certainly a small one, and I cannot give statistical results of magnitude, but in the last three years we have treated 120 cases of scarlet fever, with one death; 73 cases of diphtheria, with seven deaths; and typhoid fever, erysipelas, measles, r  theln, cerebrospinal meningitis, tuberculous meningitis, puerperal fever, tonsillitis, scarlet fever and whooping-cough, scarlet fever and diphtheria, and other diseases and combinations, quite successfully. There have been no cross-infections; very few return cases; no illness (save one attack of tonsillitis treated in the diphtheria wards) amongst the staff; no relapses or secondary rashes, and very few complications. Three cases of otitis media occurred, and were perfectly healed before discharge at the end of six weeks.

Before we commenced, in 1907, the system of bed isolation, we had quite as many troubles as others, in spite of the most careful ward separation and staff separation, but we have no apprehensions now. It is true that there are certain prime requisites for this method. One is that the matron, in the absence of a resident medical officer, should have the fullest control of her staff, and the keenest appreciation of the ideas; and in this appointment I have been fortunate. Another is that never should the bed space be less than the full minimum standard of the Local Government Board; and I hold that there is no heresy so great as supposing that children require less ward space than adults: as children they may, but as virus-holders they do not. Again, every detail of the isolation-hospital ward should be on a parity with the best surgical wards, and there must be the freest use of the sterilizer. The steam disinfecter is one sterilizer; the laundry is another, and should be worked to the utmost; but the ward sterilizers are, in small hospitals, not used as they should be.

Every patient should be kept in bed for three weeks, and, for the first few days after leaving his bed, should recline on a lounge separated by a few feet from others. We give to each one on admission a complete outfit, kept on his locker, of porcelain tray, kidney bowl, spitting mug, receiver, spray or syringe, thermometer in glass vase, glass spatula and toothbrush in glass vase, feeder, medicine glass, brush and comb, and other articles. These are all kept during the whole illness for the patient's exclusive use, and are frequently sterilized by appropriate methods, being, of course, again sterilized or, if necessary, destroyed at the end of the illness. The spatula, thermometer, and toothbrush are kept continuously in Izal solution, and the same substance shaken up with water or paroline is used for spraying, syringing, or douching

the mouth, nose, &c., every four hours during the first week, and three times a day later. No nurse passes from one prime infection to another without first disinfecting her hands and donning a special ward overall kept for the purpose, and, of course, disinfecting her hands afterwards. Certain cases of doubtful nature, and special or mixed cases are marked by a warning tab affixed to the bed card, and for each of these cases a special overall is used. In addition to the washing of the hands required from everyone on leaving a ward, disinfection for each case is required, and made easy by the placing of a stand with solution, towel, &c., at the foot of each "special" bed, or between the beds of each two or three simple cases, while rubber gloves are used in dressing wounds or attending to puerperal cases. All this means little trouble really, and is amply repaid by the knowledge that security is obtained thereby. The system can be further elaborated, but, of course, in a small place one has to go slowly and acquire material by degrees.

No doubt those who believe in inunction and swabbing with oils will say that much of this is unnecessary. No doubt it often is, for recessive cases, so far as the obvious propagation of the disease is concerned; but it is the virus from the intensive cases we want particularly to destroy, and the ritual must therefore be unhesitatingly employed. No doubt many others take just as much care as we do, and with even better results.

It is rather to the principles than to the practice that I desire to attract attention. I feel confident that if the principles are grasped we shall be able to handle our cases far more boldly than we do at present; and if, indeed, further experience justifies us in mixing our cases more freely, we shall undoubtedly require from municipal authorities less expenditure, at any rate in districts of moderate size, since not only the administration but the construction and size of isolation hospitals will be modified.

If we can show better and cheaper hospital results we shall be able to tune up to a higher pitch of efficiency and, until pathological research or clinical observation gives us more knowledge and shows us a still better way, bring scarlet fever under fuller control. I may perhaps say that, although I have to deal with a district which in 1896 had a population of 16,200, and now has one of 31,000, with all the changes that attend increase and urbanization, still the occurrence-rate of scarlet fever has gradually fallen from 3.5 per 1,000 in the quinquennium 1896-1900 to 1.6 per 1,000 in the last four years.

I am greatly obliged to you, Sir, and the members of the Section for your kind hearing. It should perhaps be said that since these notes were written I have become aware that Dr. Chapin, of Rhode Island, U.S.A., has written an essay, to which has been awarded a Fiske Memorial Prize, in which he advocates the treatment of scarlet fever, in general fever wards, on much the same lines as those I have described. I have not yet been able to obtain a copy of this essay, but am gratified to know that I am not quite alone in holding these beliefs.

I ought, also, to express my indebtedness to the valuable and stimulating paper read by Dr. Butler before the members of this Section in November, 1908.

DISCUSSION.

Dr. BUTLER said the paper had developed a view of the infectiousness of scarlet fever which had been under the consideration of the Section several times recently. That there was an intensification and recession in the activity of scarlet-fever infectiousness many observers were now agreed upon. Dr. Crookshank had gone a step further than anyone else in attempting to set a period to the extent of the intensification which occurred. But he thought there was a little ambiguity in what the author meant when he said that in about six removes the intensification had ceased, and that practically the disease had ceased to be further infectious. Dr. Crookshank said after a sequence of six cases the cycle ceased, and estimated that that would take thirty-two weeks. That meant that each successive case occurred at an interval of five weeks. But he (Dr. Butler) saw no reason for supposing that scarlet fever was going to run in a cycle with an average interval of five weeks between each secondary case to which the cases respectively gave rise. One found that scarlet fever, especially in school outbreaks, gave rise to a dropping fire of scarlet fever with intervals of a few days between each case, in which there was a range beyond the six cases which Dr. Crookshank took as the limit. The author seemed disposed to attach little importance to the view that determination of a cycle of scarlet fever was brought about by variation of susceptibility. In other words, Dr. Crookshank's thesis was that the essential factor in the spread of the disease was not so much variation in susceptibility as variation in the intensification of the poison. Against that view was the unquestionable fact that many cases of scarlet fever developed long after the exposure to infection due almost certainly to a reduction of resistance. Traumatic and surgical cases were cases in point. Only in the last few weeks a very striking instance of that came to his notice. A case of scarlet fever was notified, and in the house in which it occurred he

found that a week or two before a child had had its tonsils removed. There had been exposure of the child to scarlet fever some weeks prior to that. The child was kept under observation, and in a week it was found to be peeling. The anticipation that this child was suffering from original scarlet fever was based on the fact that children who had had adenoids or tonsils removed and had been previously exposed to scarlet fever very frequently developed the disease. The explanation of such cases was that there was a critical reduction of resistance due to the surgical operation. That variation of resistance was a determinant as important as any variation in the intensity of the infection. The author had brought under notice a fact which recently had received great prominence, that scarlet fever could be treated with other cases of disease without further extending the infection; but he felt doubtful whether those having charge of fever hospitals would be justified in putting into operation the practice which he suggested. While children were in bed, and nurses took care to avoid sources of cross-infection, cases of different diseases could be kept in the same ward. But in hospital one could not have a continuance of those conditions. There must be a stage when the patient must get up; and it was common experience that many cases contracted infection at that stage. One point touched on was the relationship between severity of attack and degree of infectivity. The more one saw of cases of scarlet fever, the more it became apparent that it was not necessarily the severe cases which were the most widely spread; and in that he agreed with the author. The cases which developed nothing but sore throat were responsible to an extraordinary degree for the spread of the disease. Its greatest infectivity was in the early stages. He thanked the author for his extremely interesting paper.

Dr. E. W. GOODALL agreed that the paper was a very bold one. Before stating any of the practical measures by which he hoped to control scarlet fever, the author entered into certain important epidemiological considerations, probably on account of the way in which they bore on his views as to the treatment of scarlet fever in and out of hospitals. With regard to the variation in the nature of scarlet fever, he said that one could not fully learn the natural history of the disease in either the hospital or the laboratory, and with that he (the speaker) agreed to some extent. But in both positions a considerable part of that natural history could be learned. The author also suggested that the disease had been modified by the interference of the public-health service, and followed that up by saying, if it was not so, the measures had been of no use at all. It was difficult to resist an argument of that kind. If one could not say, for instance, that some of the cases admitted to hospital had been saved from death, one had to admit that not much good had been done by hospital treatment in that particular direction. He (the speaker) believed lives had been saved by hospital treatment, but that was far from saying that the provision of hospitals had had much influence on the disease; indeed, the records showed that the influence of hospitals on the case-mortality had not been very great. He referred the author to a paper published about thirteen years ago by

Dr. Wilson in the *Proceedings of the Society of Medical Officers of Health*.¹ It would be worthy of any member of the Section to bring that paper up to date and see whether the deductions then drawn could be substantiated by the fuller records of notifications and deaths. Dr. Wilson concluded that hospitals had hardly any influence on the prevalence, the case-mortality or the general mortality of scarlet fever. Dr. Crookshank was very bold in stating that he *knew* he had reduced the case-mortality. In support of his contention that the natural history of scarlet fever had been modified, the author quoted Graves, because he said he could find no account of natural scarlet fever nowadays. If one took the old writers—Sydenham, Huxham, Fothergill, and, later, Trousseau and others—it would be found that scarlet fever was practically the same to-day clinically as it was any time during the past two hundred years; the only feature which had altered was the severity as shown by the mortality. Sydenham said it was hardly ever fatal, except from the officiousness of the physician. But scarlet fever had, naturally, varied very much from age to age. At present it was very mild, as it was in Sydenham's day; but there was no guarantee that in another fifty years it would not be as bad as it was in Fothergill's time. Graves, to whom Dr. Crookshank had appealed, at the very beginning of his well-known lectures on scarlet fever, particularly cautioned his readers against concluding that their treatment had anything to do with mitigating the severity of the disease. And he (Dr. Goodall) did not think there was enough evidence to justify the conclusion that there had been any real change in the disease. He thought he had seen more than the series of six cases which Dr. Crookshank mentioned, but he was barred from answering the question put by the author because he was a hospital physician, whose experience would be misleading. He (Dr. Goodall) wanted to know why the hospital specimen of scarlet fever was not a pure one; also what was meant by a pure experiment in this connexion? He thought that underlying that idea of a pure experiment was the idea of hospitalism—that patients when taken into a fever hospital necessarily developed other complications. He was prepared to admit that in some points patients brought into general fever hospitals ran some risks; but the modern idea went further—viz., that the various complications which one met with in scarlet fever were communicable from one patient to another. He was not prepared to admit that that was true to any large extent. The occurrence of complications might be, and he believed mostly was, due to the nature of the disease. He gave, some time ago, some facts in support of the view that the bulk of the patients who had complications showed them, or the causes of them, when they came in; that they did not get them in the hospital. And if that were admitted, then the cases outside the hospital were also not pure. Lastly, there was the question of the practical treatment of the disease; and on that point he asked Dr. Crookshank to give more detailed information. The hospital which Dr. Crookshank was connected with had thirty-six beds, but how were they arranged? Were there two wards

¹ *Public Health*, 1896-7, ix, appendix.

of eighteen beds each, or four wards with nine each? How many cases were in hospital at the same time? And so on. The author had apparently placed cases of certain diseases together—typhoid, erysipelas, diphtheria—and they did not spread. He (the speaker) would not expect particular diseases to spread in any carefully-managed ward even without the use of the elaborate ritual advocated by Dr. Crookshank. He asked whether Dr. Crookshank would treat measles, chicken-pox, or smallpox in the same ward as other patients. Dr. Caiger, at the South Western Hospital, would not allow measles cases in his cubicles at all, and he had found that chicken-pox spread in them. Yet these cubicles, together with a similar ritual to that advocated in the paper, afforded more isolation than that employed by Dr. Crookshank. In conclusion, while he congratulated Dr. Crookshank on his boldness, and while he agreed with him on some points, yet he felt that he must protest against certain of the deductions, because he believed they were drawn from insufficient data.

Dr. MILNE said he agreed with Dr. Crookshank in regard to the increase and decrease of scarlet fever, especially from the experience he had had long ago in the country, where cases could be watched in a way that was not possible in a hospital or city. In regard to the position which he (Dr. Milne) took up when he read his paper, and which had been that day referred to, he had received a letter from the Medical Officer of Aberdeenshire saying that the man who took his practice had never had two cases of scarlet fever in the same household when use was made of carbolic oil. A very important point, which had been referred to before, was pencil infection. That evening he had received a letter from the Bournemouth Medical Officer of Schools, saying that pharyngeal infection was found to be prevalent in one class of the school, and two children were excluded as a result of positive findings. In another case eight children were infected by one individual. The synopsis of the paper he had read referred to the cases which he had had during the twelve months from the end of October, 1898-1899, and if he had had time he would have shown how, during the past thirty years, in many private cases, and in a vast number of cases under his care, all treatment had been carried out in the way which Dr. Crookshank asked for. It was disappointing that in the matter of peeling infection some certainty could not be arrived at, and prevent medical men from holding two opposite views, some affirming that the scales were infectious and some that they were not. In any statement he had made he had left it an open question as to the source of infection. He thanked Dr. Crookshank heartily for his paper.

Dr. HAMER understood Dr. Crookshank's theorem to be that scarlet fever could only spread from case to case in the human subject for three or four removes. This theorem others, besides Dr. Crookshank, on a review of the known facts, were doubtless prepared to regard as established. But would the author proceed to draw a corollary from that, and say that the organism of scarlet fever must have an extra-human phase of existence? It seemed to him (Dr. Hamer) that the corollary must almost necessarily be drawn unless it be

decided to adopt Dr. Butler's hypothesis of the intermittent infectiousness of scarlet fever; but there were so many difficulties with regard to that. It was a very adaptable theory, and it fitted almost every individual case; but it did not explain any of the main facts of the epidemiology of the disease. He (Dr. Hamer) would, however, like further to ask Dr. Crookshank whether it was not true that in certain institutions cases of scarlet fever went on occurring sometimes for months or years. In order to shelter himself under authority, he might here refer to Dr. Niven's annual reports of some ten years ago. In the reports for the three or four years from about 1897 to 1901, Dr. Niven had given the results of his investigations in the Manchester schools with regard to the prevalence of scarlet fever. Dr. Niven referred to the "tenacity of the disease as regards particular departments" of the schools. He said: "It may go on for a year or more attacking a case in May, another in June, another in July, and so on." How did Dr. Crookshank explain that? If he decided to put on one side the question of an extra-human phase of existence of the organism, those cases repeatedly occurring in a department or class-room for months or years must represent instances of direct transference of infection from case to case. It would then be necessary to entertain the possibility, not merely of three removes, but of thirty or forty. He (Dr. Hamer) did not know whether Dr. Crookshank thought it was possible to evade this difficulty by an appeal to infection by dust, or fomites, or something of that kind. Dr. Crookshank had mentioned dusty houses, and he (Dr. Hamer) welcomed that passing reference. It seemed to him to raise very important issues. It might be either that the dust served as a mere vehicle, or, again, that it constituted the suitable nidus for an intermediate host in which a phase of existence of the scarlet-fever organism was passed.

The PRESIDENT (Dr. Niven) said that many points in the discussion were of great interest, though one would hesitate to embark on such a wide subject off-hand without previous thought. He would like to know how Dr. Crookshank would explain by his theory the main facts concerning scarlet fever. He had himself recorded instances in which the disease had begun in an indefinite manner, and had gradually gathered intensity in that manner until the cases became pronounced scarlet fever. In the same way one noticed the annual wave dying down. How did Dr. Crookshank explain the annual wave or the six-yearly wave? Every year there was an uprising of the disease in the autumn, and he did not gather that the author's theory implied that the disease passed out of the human being; and such a supposition was not necessary. Possibly there might be a succession of waves for six weeks, so that starting from the bottom of each wave, there might be another six-weeks wave. According to that, there should be a still further evolution of waves in the progress of scarlet fever. It was not very obvious how the annual wave came about. Why should there be more impulse at one time of the year than at another? There seemed nothing in the theory to account for that. It seemed to him that some new element entered into scarlet fever in the autumn;

possibly some alien infection. In return cases one got an intermixture of infection in the nostrils in the hospital; it was a hospital phenomenon. In the same way one might possibly get a mixture of infecting organisms by the action of flies, and so obtain an impulse of greater infectivity to autumnal rises which would cause cases to become more infectious; that there were new races was suggested by the different courses pursued by the different six-yearly waves. One found, time after time, that successive waves had for some reason been gradually decreasing in intensity. Probably the hospitals had much to do with that, and public-health administration still more. By isolation of a large proportion of cases in hospital, especially as the more severe cases were selected, there would be created a milder infection to operate on the community, the result being a gradual attenuation of scarlet fever. He thought that by attention to such details as had been mentioned, however trivial they might appear, one might influence the course of the disease by directing special attention to prevention at the lowest points of the annual and long waves. He would like to know how the length of the annual wave was to be explained by the theory put forth; it would take a great deal of calculation to attain the mathematical accuracy which was indicated by the reader. An important point concerned the social position of the patient. Of course, excellent results would be obtained in outlying districts; and in hospitals the results were there extremely good. The country districts supplied a better class of patients than, say, the slum districts of Manchester. The surroundings of the patients had much to do with the mortality from scarlet fever, and, in fact, the case-mortality was considerably lower among children from comparatively well-to-do quarters. The improvement in scarlet-fever mortality would thus be affiliated to the social improvement which had occurred.

Dr. CROOKSHANK, in reply, thanked the meeting for the way in which it had received his paper. He had not attempted to explain all the main facts of scarlet fever, but had been giving the society a statement of the observations he had made, and which no one had that evening contradicted, concerning intensification and attenuation; and no one had given him such a sequence as he had asked for. He had himself been trying to apply that observation to the explanation of certain facts in relation to the spread of scarlet fever, and had been dealing particularly with ordinary outbreaks of scarlet fever. In the body of his paper he had expressly left on one side milk outbreaks and sporadic cases. The following sentence in the paper was the answer to Dr. Hamer's question: "Through what phases the virus may pass, or in what media it may exist, after it is lost sight of, is matter for examination. It may perish; it may continue its generation in human beings with other or no clinical manifestations until intensification occurs." He quite realized that it was possible that the scarlet-fever virus might have a phase of existence in animals; one had reason to suppose it passed through phases in cattle; but he asked was that phase in cattle a necessary part of scarlet fever, or was it only accidental side-tracking? The other hypothesis, which prevented the hypothesis of animal phases being

a necessary corollary, was that the virus, after passing through a series of human beings, ceased to produce typical fever. He had said he saw no reason why the virus might not continue to exist in human beings; it might pass from one human being to another without causing symptoms which were recognized as those of scarlet fever. It might pass through a phase in which it produced symptoms of other illness, but he thought it more likely that the virus of scarlet fever might continue to inhabit throats of persons who were only slightly out of health without definite symptoms. Intensification would probably occur in response to variation of soil or other conditions. He had not attempted to explain why there should be a greater prevalence at one period than at another, as he was only dealing with the control of scarlet fever, not with the explanation of every fact in connexion with it. He believed that some unknown element probably did exist, which had some relation to seasons or climatic condition. More cases of erysipelas were met with in March, when the east wind prevailed; and when that wind abated, so did erysipelas. The point on which he insisted was that *eight* months was the period of seasonal prevalence; what month the period started with was not for the moment material. It was asked how the curves which occurred every six years could be explained. He suggested they might be explained by a reference to the fact that one got from year to year variations in the number of persons who were susceptible to scarlet fever, new crops of susceptible children, &c. He did not deny that there were variations in soil, but his main point was that there was a variation in the infective potency of the virus as well, and to the latter factor but little attention had been paid, though laboratory workers had repeatedly pointed out how attenuation and intensification might be induced.

In reply to the President, the social position of the patients at Mortlake had not altered for the better during the period dealt with. He had not claimed any wonderful results in connexion with the disease in Mortlake; indeed, he said in the paper that perhaps other people had had better results. With the adoption of certain methods there had been improvement; and if his methods had been radically wrong, the improvement would not have occurred. The conditions at Barnes and Mortlake had, indeed, been somewhat disadvantageous. Some eight years ago the district was rural and the houses were widely separated, but in the period of his observation there had been a doubling of the population and such an increase in building that rows of small houses had been built, with attendant overcrowding of elementary schools, &c. He did not think that Dr. Butler and he differed much from each other. If one took a series of six cases, and assumed infection in each case to occur at the end of five or six weeks, the whole sequence would take about thirty-two weeks, and that was the probable maximum of time over which the infection would spread. There might, of course, be a series in which infection occurred in each case at the end of eight or nine weeks. He thought the extremities of the curves, if plotted out, would be much as in the diagrams given in Sir Clifford Allbutt's "*System of Medicine*."¹ The most abrupt rise was during the central six weeks, when there

¹ "*System of Medicine*," 1906, ii, part 1, pp. 410-75.

was a tendency to be the greatest accumulation of cases. An apparently long series of cases might continue because of the intervention of fresh infection. The question of exhaustion of soil and susceptibility he had already mentioned, and he did not deny its importance, but he was calling attention to another factor. He was glad Dr. Butler mentioned the tonsil cases, because he believed the reason why children who had their tonsils removed so often contracted scarlet fever was that such a large breach of surface was created. It was true that Dr. Goodall had knocked down some controversial ninepins, but he had himself set them up. He (Dr. Crookshank) had no idea of giving offence to hospital superintendents. The fact remained, however, that the history of scarlet fever in isolation hospitals was not the natural history of the disease, but the history of the disease under artificial conditions. Another ninepin which Dr. Goodall set up was the question of hospital administration having modified the natural history of scarlet fever. He contended that public-health administration had so modified it, but this and hospital administration were not synonymous terms. By public-health administration he meant disinfection, isolation at patients' homes, the cutting down of school cases, &c. He thought, however, that hospital treatment had tended to modify the case-mortality of scarlet fever, and the tables in Allbutt's System showed how the case-mortality had continuously fallen in London during the last thirty or forty years. No one would deny that scarlet fever was better treated in hospitals than outside, and that, other things being equal, the mortality was less in hospital than in the slums. If the explanation of the differences which he had given was not the correct one, he would be glad to hear another. Dr. Goodall made light of his (Dr. Crookshank's) remark that Graves' was the last vivid account of scarlet fever in the natural state, but Dr. Goodall did not supply a more recent one. He did not know any part of England where scarlet fever occurred now with the raging intensity described by Graves. He believed that the natural history of scarlet fever had been modified of late years. There were no hospital return cases in the time of Graves. He certainly drew a comparison between the epidemics in those days and the seasonal prevalence in London, but because two groups of facts had points of difference was no reason why there should not also be points of resemblance. With regard to the question of the purity of the isolation-hospital experiment, hospital cases could not be pure ones. If a child went home and took scarlet fever to its brother or sister at home, and that brother came back as a return case, it did not follow that the return case was infected with the virus which the first case had been infected with. It was possible that the first child was imperfectly immunized and took home another strain of infection from the hospital. When a doctor had several cases in a family under observation, and that family was isolated privately, it was a much purer experiment than was the hospital experiment. He agreed that cases went into hospital with impure infections. Relapses were far more common in hospital than outside; he regarded relapses as infections of imperfectly immunized patients. When he took charge of the isolation hospital at Mortlake it was inadequate to the needs of the district.

He had to deal with only two blocks, and owing to the excessive prevalence of the disease there they were faced with difficulties; diphtheria in one block, scarlet fever in another, and no observation wards. He did his best to keep the diseases separate by having one staff of nurses for one block and one for another. But, in spite of that, there were the usual cross-infections. They set to work to devise some means of preventing cross-infections, and by carrying out the ritual they found they got along very well indeed. The patients themselves did very much better, because there were not so many secondary rises of temperature or rashes or enlarged glands, &c. The ritual was nothing more than the application of the methods of asepsis to medical wards. Every surgeon would admit that in country places people did very well in spite of lack of aseptic precautions, but every now and then disaster occurred, and if disasters were to be prevented the necessary ritual must be observed. His point had been as to whether fever cases could be safely treated side by side. He had not deliberately put them side by side, as he felt limited by a sense of responsibility. But if there was a consensus of professional opinion in favour of dealing with such cases more boldly, he would be glad to go further. He had not referred to cases of variola or varicella, as he had been dealing with throat and nose infections; but in Berlin cases of variola were treated successfully in general hospitals, though, of course, vaccination was enforced.

Epidemiological Section.

February 25, 1910.

Dr. JAMES NIVEN, President of the Section, in the Chair.

The Communication of Diarrhœa from the Sick to the Healthy.

By J. E. SANDILANDS, M.D.

THAT diarrhœa is an infectious disease capable of spreading from the sick to the healthy is now assumed by a large section of the medical community, and the principal object of this paper is to consider how far this assumption is in accordance with the facts observed in the course of an investigation into the deaths from diarrhœa which took place in Kensington during the third and fourth quarters of the year 1909.

In Leicester, during the epidemic seasons of 1881 and 1882, Dr. Ballard (1887-8, p. 14) and Mr. Power made personal visits to the houses where deaths from diarrhœa occurred, and, although in this way they collected detailed records of no fewer than 340 fatal cases, they do not appear to have obtained any important evidence that the disease was communicable. In one of the few scanty references to infection contained in his report to the Local Government Board, Ballard (p. 18) says: "In proof of the occasional communicability of an epidemic diarrhœa, I [append] . . . a report by Dr. Low on several occurrences of the sort. . . . Other apparent occurrences of this kind have come to my knowledge, but this is not the place to consider the subject further." The wording of the last sentence suggests that these other apparent occurrences were not within the writer's own personal experience, and Dr. Low's cases remain as the only evidence of communicability in a report which was destined never to be completed.

The Helmsley cases described by Dr. Low (1887, p. 127) consist of the following four groups: Group I, 10 cases occurring in May, 1876; Group II, 14 cases occurring in February, 1880; Group III, 60 cases occurring in December, 1882; Group IV, 62 cases occurring in September, 1886. It will be noted that the first three groups did not occur in the diarrhœa season, whilst two of them occurred in the depth of winter. Group III was anomalous, not only in its seasonal incidence, but from the fact that certain adult patients passed bloody stools, and in two adults the looseness of the bowels continued for several weeks with occasional passing of blood in the motions. The fourth group occurred in the diarrhœa season, but should probably be reckoned as anomalous on account of the astoundingly contagious properties displayed by the fæcal discharges of the sick. Thus: "One small boy . . . unfortunately messed his breeches in school, and shortly afterwards at least eight of the school children, who were present in the room when this accident occurred, were taken ill." Of a girl named Ashpole it is said: ". . . this is the only case out of the 60 which followed which could not be *clearly* traced to exposure to infection from diarrhœal discharges." It is also related how a labourer's wife with her little daughter was passing an infected cottage when a child came out and threw a pot full of liquid excrement on to a dung-heap close to the path. This woman and her child were simultaneously taken ill the same evening with violent sickness and purging, and in two days the rest of the family were also ill. In this case, as in many of the others described by Dr. Low, the incubation period was less than twenty-four hours.

In the absence of any record of similar experiences among the 340 Leicester cases, it is perhaps well that summer diarrhœa should not be lightly assumed to possess infectious properties, and that Dr. Low's records of certain exceptional epidemics should not be hastily accepted as having any direct or necessary bearing on the causation of diarrhœa in other places and in other years. We have, in fact, to consider not only whether diarrhœa is *capable* of spreading from the sick to the healthy, but also whether it *does* so spread in the epidemics which recur annually throughout the country. That it is *capable* of spreading in this manner is suggested by the experiments recently made by Metchnikoff (1909, p. 1649):—

"(1) A chimpanzee . . . in good health swallowed a small quantity of the green stools of a girl, aged 6 months, suffering from acute gastro-enteritis. The following day the chimpanzee began to suffer from a very watery diarrhœa. . . .

"(2) A second chimpanzee swallowed a small quantity of fæcal matter from the first chimpanzee. On the following day diarrhœa appeared, which lasted ten days.

"(3) Two older chimpanzees . . . were given a small quantity of fæcal matter from the same infant. One developed diarrhœa the next day; the other chimpanzee remained normal for ten days, and then had diarrhœa, which only lasted one day. With laboratory evidence might also be classed the experience of Dr. W. Johnston (1878, p. 283), who suffered from three attacks of severe diarrhœa during a prolonged microscopical investigation of the dejecta of diarrhœa patients."¹

If Metchnikoff's experiment be provisionally accepted as indicating that the stools of affected infants contain the causative agent of diarrhœa, we must admit the possibility of infection being conveyed in diarrhœal excrement by flies from house to house over considerable distances.

Newsholme (1903, p. 21) has pointed out that food in the houses of the poor becomes contaminated by flies which may have come from " . . . the liquid stools of a diarrhœal patient in a neighbouring house," and Dr. Davies has found that diarrhœa has a tendency to spread from the back of houses in one street to the adjoining backs of the next street. He states that this is what would be expected on the hypothesis that the infection was conveyed by flies.

The hypothesis of house-to-house infection by these means is attractive, but could hardly be upheld in the absence of valid evidence of the spread of diarrhœa from family to family in the same house, and more especially from person to person in the same family. That the collection of such evidence may involve considerable labour is to be gathered from the fact that out of 268 cases of diarrhœa investigated by Dr. Davies (1908, p. 4), only 32, or 12 per cent., were attributed to infection derived from other members of the same household, whilst 29 were attributed to infection from persons living in neighbouring houses.

In the Annual Report on the Health of Winchester for 1906 (p. 31) the following series of cases of diarrhœa were published. They occurred in two houses in a single row of eighteen cottages, and are described as of short duration, of considerable severity, and as accompanied by vomiting. Two months before the investigation was made the death of an infant at the cottage numbered 5 in the same row had been certified as due to enteritis.

¹ The reference to Dr. W. Johnston's paper in the *Lancet* of 1878 will be found in "Longstaff's Studies in Statistics," p. 283.

WINCHESTER CASES.

Address	Date of onset	Number of cases
No. 12 	{ October 5 	1 adult
	{ October 23 	3 children
	{ October 27 	1 child
	{ November 3 	1 adult
No. 11 	{ October 27 	2 adults
	{ October 30 	1 child

These cases are open to the same objection as those published by Dr. Low, in that they constitute an isolated outbreak of a possibly infectious diarrhœa in a particular row of cottages, and do not necessarily belong to the same class as the fatal cases in infants which furnish the annual mortality waves of epidemic diarrhœa.

The cases investigated in Kensington are not open to criticism on the same score, since the nature of the inquiry was determined beforehand in June and was not concerned with an existing epidemic in a given neighbourhood, but designed with the object of obtaining information with regard to the fatal cases of diarrhœa which it was assumed would occur in Kensington before the end of the year. Particulars were obtained as to 35 cases distributed among thirty-five houses which, with two exceptions, were tenement houses occupied by several families of the poorer class. The records of 28 cases are based on my own observations; notes on the remaining 7 cases were made by a sanitary inspector. In sixteen of the houses visited, in addition to the deaths registered as due to diarrhœa, 1 or more non-fatal cases had occurred either in the same family or in another family in the same house. Three instances of multiple invasion reported in the first instance to the sanitary inspector were verified by subsequent inquiries; the records of the remaining 13 examples of multiple invasion represent the results of my own investigations. The total number of fatal and non-fatal cases occurring in the thirty-five houses where deaths had been registered as due to diarrhœa was 64, and the total number of families visited was ninety-two. The following table presents in a summary form the scope and limitations of the inquiry:—

KENSINGTON CASES OF DIARRHŒA.

Cases investigated 	64
Number of fatal cases 	35
Houses where fatal cases occurred 	35
Families where fatal cases occurred 	35
Families showing multiple invasion 	12
Houses showing multiple invasion 	16
Families (or tenements) visited 	92

Reference has already been made to the occurrence of multiple cases in certain families and houses, and before proceeding further it will be convenient to assess the significance which we attach to the phenomenon of multiple invasion and the other characteristics commonly displayed by infectious diseases.

In his Report on Epidemic Pleuro-Pneumonia, Ballard (1888-9, p. 163) commented on the characteristic features of communicable diseases in the following words:—

“(1) *Epidemicity*, frequent or only occasional, is one of the phenomena which characterise most of the communicable diseases.

“(2) *The occurrence of multiple cases* in one house or family. Such an event indeed is no evidence of the communication of a given disease from the sick to the healthy, when standing alone as an exceptional occurrence; though it may have some significance in this direction when of frequent occurrence in the course of an epidemic. The reason for hesitating to accept infection from person to person in either of the above cases is that it might be that all persons attacked had been exposed to the influence of one and the same extraneous cause, living as they would have been under the same or similar conditions and surroundings. There is, of course, direct suggestion of infection from person to person when all the members of a household are attacked not simultaneously or nearly so, but more or less consecutively.

“(3) *Communicable diseases* spread most readily under circumstances . . . which conduce to the closest association of the sick and the healthy. One of these circumstances is aggregation upon a limited area . . . another is crowding together of persons within dwellings.”

To these characteristics should certainly be added the occurrence of any given disease outside its normal environment among the inmates of hospitals to which infected persons have been removed, neither will it be necessary to refer here by way of illustration to the importance of the part played by hospital experience in establishing the contagious nature of typhoid fever and the non-contagious properties of plague. Put more briefly, the characteristics of communicable diseases are:—

- (1) Epidemicity.
- (2) Multiple invasion of houses or families.
- (3) Consecutive dates of onset in multiple cases.
- (4) Incidence on crowded areas or dwellings.
- (5) Incidence on those in contact with the sick in hospital.

In the light of these five indications of communicability the circumstances surrounding the fatal cases of diarrhoea which were registered in Kensington in the four months July to October, 1909, may now be reviewed.

(1) EPIDEMICITY.

Fig. 1 represents the fortnightly incidence of diarrhoea in Kensington and London. The curves, which closely correspond in form and time, indicate the epidemic character of the outbreaks in both places. The curve for Kensington is based on the dates of onset in fatal cases of diarrhoea; the apparent correspondence in time of the London curve,

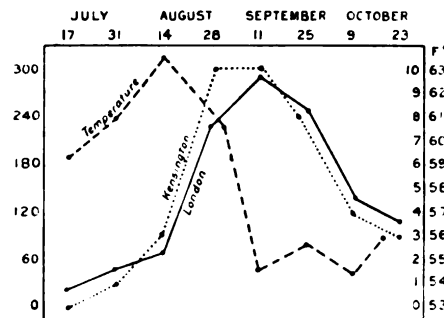


Fig. 1. Deaths from Diarrhoea in London during the fortnightly periods ending on the dates indicated in the chart and fatal attacks of Diarrhoea in Kensington beginning during the same fortnightly periods. The chart also shows the Mean Temperature of the Air from July 3 to October 23 in each fortnight.

which is based on dates of death, must therefore be taken as indicating that the outbreak of diarrhoea in London began some fourteen days before the outbreak in Kensington.

(2) MULTIPLE INVASION OF HOUSES AND FAMILIES.

Details as to the multiple cases, which occurred in sixteen of the houses and twelve of the families visited, are given in Table I.

Since the phenomenon of multiple invasion carries little weight unless it is of frequent occurrence, it should be noted that multiple cases were only recorded in sixteen out of thirty-five houses, and twelve out of forty-three families attacked. From the figures in Table II it will be understood that the eight families attacked by non-fatal diarrhoea all lived in houses where fatal diarrhoea had occurred in other tenements.

TABLE I.—DATES OF ONSET OF CASES OF DIARRHŒA IN HOUSES AND FAMILIES EXPERIENCING MULTIPLE INVASION.

House No.	Family surname	Sex	Age (years)	Date of onset	Date of death or recovery	Remarks
+ 1	B	F.	1 $\frac{2}{12}$	August 1	Sept. 11, R.	Taken daily into A's rooms.
	A	M.	1	August 21	Sept. 10, D.	Contact with B, <i>vide</i> above.
2	A	F.	1 $\frac{4}{12}$	August 13	Aug. 15, D.	—
	B	M.	10	August 21	— R.	No proof of contact.
3	A	M.	1 $\frac{4}{12}$	August 13	Aug. 20, D.	New milk; no proof of contact.
	B	F.	1 $\frac{1}{2}$	August 22	— R.	"Star" condensed milk.
4	A	F.	2	August 13	Aug. 17, R.	Severe diarrhœa.
	A	F.	8 $\frac{1}{2}$	August 17	Aug. 21, D.	Healthy, "fat as a mole" till onset.
	B	F.	25+	August 22	— R.	Severe diarrhœa, with vomiting; denies contact.
+ 5	B	M.	6 $\frac{1}{12}$	Sept. 1	Sept. 3, R.	Left the house Sept. 3, very ill.
	A	F.	1 $\frac{1}{2}$	Sept. 8	Sept. 12, D.	Incubation five days; contact not proved.
+ 6	A	F.	1 $\frac{4}{12}$	Sept. 1	Sept. 6, D.	Nestlé's condensed milk.
	A	M.	2	Sept. 8	— R.	New milk, no condensed.
7	A	M.	1 $\frac{4}{12}$	Sept. 6	Sept. 23, D.	"Fine, healthy child" before attack; families A and B on same floor, model dwellings; wash-house and water-closet in common.
	A	F.	25+	Sept. 16	— R.	
	B	F.	10	Sept. 21	— R.	
	B	M.	3	Sept. 21	— R.	
	A	M.	4	Sept. 24	— R.	
8	B	F.	25+	Sept. 9	Sept. 29, R.	Played with baby A.
	A	F.	8 $\frac{1}{12}$	Sept. 24	Sept. 27, D.	Contact with B.
	A	F.	50+	October 1	— R.	Severe, with vomiting.
9	B	M.	2	Sept. 1	Sept. 14, R.	Baby A minded during day in B's room; both B children diagnosed, St. Mary's Hospital, as "diarrhœa and vomiting."
	B	F.	1 $\frac{2}{12}$	Sept. 6	— R.	
	A	F.	2 $\frac{1}{2}$	Sept. 5	Sept. 29, D.	
10	A	F.	6	Sept. 1	— R.	—
	A	F.	6 $\frac{1}{12}$	Sept. 12	Oct. 7, D.	—
	A	F.	2	Sept. 12	Sept. 19, R.	Diarrhœa and vomiting.
11	A	F.	6 $\frac{1}{12}$	Sept. 16	Oct. 11, D.	Nestlé's milk } Taken to hospital
	B	F.	1 $\frac{1}{2}$	Sept. 16	— R.	New milk } together same day.
12	A	F.	2	Sept. 26	— R.	A mews; father and mother had severe diarrhœa, same house, last year.
	A	M.	4	Sept. 28	Sept. 30, D.	
13	A	M.	8 $\frac{1}{12}$	October 5	Oct. 13, D.	Wealthy family, large house.
	A	F.	35+	October 17	— R.	Incubation four days +.
14	A	M.	5	October 8	— R.	Children, "Goat" brand condensed milk in tea, no new milk; baby, healthy and strong before attack, new milk only.
	A	F.	4	October 8	— R.	
	A	F.	30+	October 8	— R.	
	A	F.	2 $\frac{1}{12}$	October 15	Oct. 16, D.	
15	A	M.	2	October 15	— R.	Attended hospital.
	A	M.	1 $\frac{2}{12}$	October 22	Oct. 24, D.	"A fine, healthy boy."
16	B	F.	60+	October 20	— R.	Severe retching with diarrhœa.
	B	F.	30+	October 24	— R.	Diarrhœa, no vomiting.
	B	M.	8 $\frac{1}{12}$	October 25	— R.	
	B	—	—	—	— R.	
	B	—	—	—	— R.	
	B	F.	7	October 30	— R.	Four children, aged 8 months to 7 years, taken ill "one after another"; not simultaneously.
	A	M.	3 $\frac{1}{12}$	October 29	Oct. 31, D.	Nestlé's condensed milk.

The houses invaded are designated by the numbers in the first column. Members of the same family are designated by the name-letter A or B. † These three cases were investigated in the first instance by Miss de Chaumont, Lady Sanitary Inspector.

TABLE II.—MULTIPLE CASES AMONG PERSONS WHO RESIDED IN HOUSES WHERE DEATHS FROM DIARRHŒA OCCURRED.

	Number attacked	Number showing multiple cases
Houses where fatal diarrhœa occurred ...	35	16
Families where fatal diarrhœa occurred ...	35	9
Families attacked by non-fatal diarrhœa in 35 houses ...	8	3
Total families attacked by fatal or non-fatal diarrhœa in 35 houses where deaths occurred ...	43	12

In only seven houses and four families showing multiple invasion did the number of persons attacked exceed two.

(3) CONSECUTIVE DATES OF ONSET IN MULTIPLE CASES.

The conditions to be satisfied under this head are that all the members of a household should be attacked not simultaneously or nearly so, but more or less consecutively. The table of cases sets forth the records of twelve families in which multiple cases of diarrhœa occurred. In eleven of these families a period of the average duration of eight days intervened between the dates of onset of the primary and secondary cases. In one instance only was the interval as short as two days, in the remaining ten families the interval was four days or more, but did not in any case exceed three weeks. The intervals between the dates of onset are not to be taken as necessarily representing incubation periods, since they are bridged in every instance by the continued illness of the primary case.

The condition that *all* the members of a household should be attacked constitutes a very severe test of communicability, and was by no means fulfilled, since two families only showed a sequence of more than two cases. It should, however, be borne in mind that sequences in highly infectious diseases, such as measles and whooping-cough, are necessarily limited to small numbers in families where the older children are protected by previous attack, and may, for similar reasons, be limited in families attacked by diarrhœa.

The phenomenon of simultaneous onset in the primary cases was only observed in two out of the twelve families in which multiple cases occurred. In one instance three persons, aged 4, 5, and 30, were attacked on or about the same day; in the other instance simultaneous onset was recorded in two children aged 3 and 10. A single example of simultaneous onset in two secondary cases may be ignored,

since both may reasonably be attributed to simultaneous infection by the primary case.

When we substitute the tenement house for the family as the unit experiencing multiple invasion we come to more doubtful ground, and are obliged to beg the essential question of effective contact. In nearly every house the yard, washhouse, w.c., the passages and the staircases were used in common by members of the families attacked. In a few instances the patient is known to have visited the family in which secondary cases occurred; in the majority of cases casual contact in the same room is denied or not proved. The order of attack in the houses where multiple cases occurred is shown in Table III.

TABLE III.—ORDER OF ATTACK IN PERSONS LIVING IN TENEMENT HOUSES WHERE MULTIPLE CASES OF DIARRHŒA OCCURRED.

Simultaneous onset in primary cases	2 houses
Onset of cases not simultaneous	14 „
Simultaneous onset in secondary cases	3 „
Sequence of more than two cases	4 „
Average interval between primary and secondary cases	8 days

In only one instance did the secondary case succeed the primary within four days; the usual interval varied between four and nine days; longer intervals, of which three weeks was the maximum period recorded, were bridged by continued illness in the primary case. Whether houses or families are considered, the facts recorded demonstrate that simultaneous onset was rare, that consecutive onset was the rule, but that only one instance (Case 16) has been brought forward where *all* the members of a family were attacked not simultaneously or nearly so, but more or less consecutively.

The Manchester Cases.

At the last moment, when the proofs of this paper had already been printed, my attention was called to the valuable study of the ætiology of diarrhœa published by Dr. Niven in 1905. During the summer of 1904 Dr. Niven caused the most exhaustive inquiries to be made into all the circumstances attending the occurrence of 111 fatal cases of diarrhœa in infants, especial attention being paid to phenomena which suggested that the disease was communicable. In respect to three times the number of cases, the records obtained include the whole of the ground which has been covered in this paper.

Of 111 families where deaths from diarrhœa occurred, 36 appear to have experienced multiple invasion. In 31 of these families the number of multiple cases was limited to two, and only four families showed sequences of three or more cases. Two of the families in which more than two cases occurred were

attacked by a disease which may have been enteric fever. In multiple cases the dates of onset are consecutive, and no instance of simultaneous onset is quoted. Some 50 instances of the communication of diarrhœa by contact from sick to healthy persons were recorded, and in each instance the circumstances are given in detail. Obviously evidence of this nature cannot be summarized, and each example of apparent infection must be judged in the light of the histories which will be found in Dr. Niven's report.

The experience of the Manchester families in which fatal diarrhœa occurred may be represented in tabular form, as follows :—

Families where fatal diarrhœa occurred ¹	111
Families experiencing multiple invasion ¹	36
Simultaneous onset in primary cases	<i>Nil</i>
Simultaneous onset in secondary cases	<i>Nil</i>
Sequences of more than two cases ¹	4

Precise dates of onset are not given in every instance, so that the average interval which elapsed between the occurrence of the primary and secondary cases cannot be stated.

(4) THE INFLUENCE OF AGGREGATION.

With two exceptions, the fatal cases of diarrhœa which occurred in Kensington were strictly limited to crowded tenement houses in crowded areas. On the other hand, Newsholme (1899, p. 152) has proved conclusively that when one town is compared with another, the mere density of population bears no proportion to diarrhœal mortality. There is, however, no suggestion that the incidence of diarrhœa is not greater on the more thickly populated districts of the same town, as may be gathered from Dr. Newsholme's statement (p. 153) that "epidemic diarrhœa . . . is a disease of the . . . lower labouring classes to a preponderant extent"—that is to say, of the classes who live in crowded quarters. It is also in England a disease of urban communities, though this rule does not hold good in Malta, where the diarrhœal mortality is very much greater in the rural districts. Dr. Critien (1909, p. 333), the Medical Officer of Health for Malta, in a report of great interest, has drawn attention to the steady decline in the urban diarrhœal death-rate which has followed the provision of a water-carriage system of sewerage in the towns.

TABLE IV.—NUMBER OF DEATHS UNDER 1 YEAR FROM DIARRHŒA PER 1,000 BIRTHS IN MALTESE TOWNS AND VILLAGES.

Districts	Population	1901	1902-3	1903-4	1904-5
Urban	53,168	90	86	86	78
Rural	53,017	102	96	120	121

¹ Two of these families were attacked by a disease which may have been enteric fever.

In the villages diarrhoeal excreta are described as lying on the road-way, and faecal matter of human origin, in the absence of water carriage, is, no doubt, disposed of in such a manner as to be freely exposed. In further explanation of this reversal of the urban and rural death-rates experienced in England, it should be noted that the overcrowding on space, as well as in dwellings in the Maltese villages, is phenomenal, the least crowded of the villages containing more than thirty persons per acre.

In summing up the question of aggregation it may be said that in Kensington the heaviest incidence of diarrhoea has in the last year been on the most crowded neighbourhoods, and that a similar incidence is no doubt the rule in other urban communities.

(5) INCIDENCE ON THOSE IN CONTACT WITH THE SICK IN HOSPITALS.

Owing to the courtesy of the resident medical officers, I have been able to obtain information from thirteen of the principal children's hospitals in the country as to the results which follow the admission into the general wards of infants suffering from zymotic enteritis. A summary of the information obtained will be found in Table V.

TABLE V.—EXPERIENCE OF 13 CHILDREN'S HOSPITALS AS TO THE INFECTION OF CHILDREN AND NURSES BY PATIENTS SUFFERING FROM DIARRHOEA.

Alleged infection of nurses	4 hospitals
Alleged infection of patients	7 „
Nurses not attacked	9 „
Patients not attacked	5 „
Children with diarrhoea isolated	1 hospital

In four hospitals there is some evidence suggesting that nurses in charge of patients suffering from diarrhoea have themselves become infected with the same complaint. Dates of onset and other details are lacking.

(1) From one hospital comes the information that “three years ago both nurses and patients admitted for some other ailment suffered from diarrhoea at a time when large numbers of cases of diarrhoea had been admitted into the general wards.”

(2) The resident medical officer of a second hospital had a special ward of six beds for the treatment of zymotic enteritis. “The three nurses who did eight-hour stretches all had more or less severe diarrhoea and vomiting. None of the other nurses were attacked, and no case of diarrhoea and vomiting was brought into the other wards.”

(3) In a third hospital Morgan (1906, p. 911) succeeded in isolating the bacillus known as Morgan's bacillus No. 1 from the stools of a nurse

"who had contracted diarrhoea from a patient in the ward set apart for this disease." Batten (1906, p. 177), in a paper published in the *Clinical Journal*, describes this nurse's attack as severe and accompanied by vomiting, and adds "that three other nurses suffered from mild attacks which may or may not have been so contracted."

(4) From a fourth hospital the medical officer writes as follows: "I can only hear of one nurse who had an attack of diarrhoea while nursing these patients, and who is not subject to such attacks. There appears to have been no case of diarrhoea among nurses in the other wards. I am told that in 1908 there was a similar epidemic, and that then also one nurse had an attack of diarrhoea while in attendance on the diarrhoea cases."

In the remaining nine hospitals there has been no evidence of the spread of diarrhoea from patient to nurse. In some cases the immunity of the nurses is attributed to the precautions taken to prevent infection, but from the answers received it may be safely inferred that in these hospitals severe diarrhoea among the nursing staff, if it ever occurs, is extremely rare.

INFECTION OF PATIENTS IN HOSPITAL.

In reply to the question as to whether the admission of cases of diarrhoea into general wards had apparently given rise to attacks of diarrhoea in patients under treatment for some other complaint, the medical officers of seven hospitals wrote stating that this phenomenon had occurred within their own experience. The information received is, with one exception, in general terms, and details are lacking. One answer already quoted refers to a particular outbreak three years ago, and the following reply may serve as an example of others: "Our experience as regards the nursing staff is directly opposed to any infection from children suffering from zymotic enteritis. It, however, frequently happens that if children suffering from that disease are introduced into wards containing unaffected children, the latter develop it, and in an extremely acute form. Not, however, the older children, but as a rule the babies under one year old."

The following table of cases has been sent me from a children's hospital by the resident medical officer, to whom I am very much indebted for the valuable material it contains. Wards VIII and IX, in which cases occurred, are in separate buildings, but are administered by the same nursing staff.

TABLE VI.—CHILDREN ALLEGED TO HAVE ACQUIRED DIARRHŒA IN HOSPITAL FROM OTHER PATIENTS, MARKED *, WITH DATE IN ITALICS. THOSE ADMITTED WITH DIARRHŒA ARE MARKED †.

Case No.	Age (years)	Disease on Admission	Ward No.	Date of admission	DIARRHŒA (DATES OF)	
					Occurrence in hospital	Recovery or death
1	† 2	Diarrhœa	VIII	July 19	July 19	August 10, R.
2	† $\frac{2}{13}$	Marasmus and diarrhœa	„	August 13	August 13	August 27, D.
3	* $\frac{4}{13}$	Pneumonia	„	July 29	<i>August 19</i>	Sept. 22, D.
4	† $\frac{2}{13}$	Gastro-enteritis	„	August 30	August 30	Sept. 24, D.
5	† $\frac{6}{13}$	Marasmus and diarrhœa	„	Sept. 3	Sept. 3	Sept. 5, D.
6	† $\frac{1}{13}$	Gastro-enteritis, meningitis	„	Sept. 6	Sept. 6	Sept. 24, D.
7	* $\frac{1}{12}$	Congenital heart disease and marasmus	„	August 27	<i>Sept. 11</i>	Sept. 20, D.
8	* 1	Pneumonia	„	Sept. 7	<i>Sept. 11</i>	Sept. 25, R.
9	* $\frac{1}{13}$	Pneumonia	„	Sept. 12	<i>Sept. 16</i>	Sept. 24, R.
10	† $\frac{1}{13}$	Gastro-enteritis	„	Sept. 21	Sept. 21	Oct. 12, D.
11	† $\frac{1}{12}$	Gastro-enteritis	„	October 14	October 14	Oct. 19, D.
12	? $\frac{1}{12}$	Congenital heart disease	IX	July 30	<i>October 17</i>	Oct. 19, D.
13	? $\frac{5}{13}$	Pneumonia	„	October 12	<i>October 19</i>	Oct. 27, D.
14	† $\frac{10}{13}$	Enteritis	„	August 11	August 11	August 11, D.
15	1	Rickets and gastro-enteritis	„	July 29	August 12	Sept. 3, R.
16	* $\frac{6}{13}$	Pneumonia	„	August 12	<i>August 28</i>	Sept. 2, D.

The table certainly suggests very strongly that four children, Cases 3, 8, 9 and 16, admitted with pneumonia, became infected with diarrhœa by patients in the same ward who were under treatment for diarrhœa or gastro-enteritis. Of the 5 cases marked with an asterisk the medical officer in charge says: "I am convinced that these 5 cases were infected from other patients, although every precaution was taken to prevent infection, the diarrhœa cases being treated practically like typhoid cases."

In five hospitals there has been no experience of the spread of diarrhœa from patient to patient, and Dr. Ralph Vincent, with his unique experience in the Infants' Hospital in Vincent's Square, holds very strongly the opinion that diarrhœa is not a communicable disease. In the last week of October, 1909, Dr. Vincent wrote to me as follows: "At the times when the disease is prevalent, numerous cases of zymotic enteritis are admitted, mostly of an extremely severe type. No isolation of the cases is attempted: they lie in cots side by side with infants who are suffering from the other nutritional diseases. No infant has ever contracted 'zymotic enteritis' in the hospital." In this institution the duty of feeding the patients and dealing with the excreta, both of diarrhœa cases and other infants, is entrusted to the same nurse, who

takes no special precautions to prevent infection, and is instructed in general terms only to be cleanly in her work.

Whilst it is true that Dr. Vincent holds diarrhœa to be a non-communicable disease, elaborate precautions are nevertheless taken to insure the purity of the milk given to the babies, and, although these measures are directed on general lines against contamination of any kind whatsoever, they have the incidental effect of protecting the milk from the introduction of infectious matter from the wards. The feeds for patients are prepared by a special staff of women in a laboratory provided for the purpose. The milk for each child is contained in a stoppered bottle, which does not leave the laboratory and is not entrusted to the ward nurse until she is about to feed her patient. It is her duty on receiving the sealed bottle containing the feed to place it in hot water, to remove the stopper, and to insert a thermometer into the milk. When the milk has risen to the requisite temperature, she removes the thermometer, fits a sterilized teat on to the mouth of the bottle, and administers the feed to the infant in her charge. The food which is given to these infants may thus be said to be as effectually protected from specific contamination as breast milk, except for the momentary risk which in theory might attend the insertion of the thermometer.

In summarizing the experience of children's hospitals, the evidence may be described as conflicting and inconclusive; but, when this admission has been made, it may with advantage be qualified by reference to the controversy as to the infectious nature of typhoid fever which formerly divided epidemiologists into two camps.

In his book on continued fevers, Murchison states that during nine years 3,555 cases of enteric fever were treated in the London Fever Hospital, along with 5,144 patients not suffering from any specific fever, and yet not one of the latter contracted typhoid. In the case of enteric fever the positive evidence of a comparatively small number of instances of communicability has rightly been held to outweigh this mass of negative experience; and so with diarrhœa it may not be unreasonable to ask that a certain weight should be attached to examples of apparent case-to-case infection, even though they be few in number and directly opposed to the experience of certain hospitals.

DETAILS OF CERTAIN CASES IN HOUSES WHERE DEATHS OCCURRED.

The circumstances attending Case 9, in the table of cases of multiple invasion, were as follows: On September 1 a boy, aged 2, developed

diarrhoea and attended as an out-patient at St. Mary's Hospital. On September 6 his baby-sister was seized with diarrhoea and vomiting. On the previous day, September 5, the baby of a woman in the next room developed an attack of diarrhoea, which ended fatally on September 29. Since the middle of August this child had been left during the whole day with the family in which the boy was taken ill on September 1. The secondary cases appear to have been infected simultaneously by the boy aged 2. One was fed on cow's milk, the other on Nestlé's milk.

In Case 14 two young children and their mother developed diarrhoea on the same date, October 8. They took condensed milk in tea, but no cow's milk. On October 15 the baby, aged 2 months—a healthy, strong child—was seized with diarrhoea and died within forty-eight hours. It was fed on the breast and occasionally given a little new milk. The little girl, aged 4, was said to have not only dirtied the bed, but also the stairs during her illness.

Case 13 is of interest because it did not occur in the usual environment, but in a large house in one of the best parts of Kensington. A baby, aged 8 months, was taken ill with diarrhoea on October 5, and died on October 13. Five days later a lady, who had removed the soiled napkins from the sick room and washed them, was seized with violent diarrhoea.

In Case 16 a woman, aged about 55 or 60, living in a basement with her son's family, developed diarrhoea on October 20. On October 24 the mother of the family fell ill. Her four children, including a baby, aged 8 months, developed diarrhoea within the next few days. The baby was fed on the breast with a little beef tea, but no cow's milk. A child, aged 3 months, living on the ground floor of the same house, fell ill with diarrhoea on October 29 (five days after the second case in the basement) and died within three days. This child was fed on condensed milk; contact with the family in the basement was denied.

DETAILS OF CASES IN HOUSES WHERE NO DEATHS OCCURRED.

The cases of diarrhoea mentioned in Table VII, to which no reference has hitherto been made in this paper, occurred in two houses where none of the inmates acquired the disease in a fatal form.

In Case 1 a baby, aged 16 months, living on the ground floor, was attacked with profuse diarrhoea on September 10, and was treated by a medical man on September 17. A father and a child aged 2, living on the third floor in the same house, fell ill with diarrhoea on September 23.

The attack in the father was accompanied by vomiting, and he was so bad that his wife does not know how he continued to work. She herself was taken ill on September 30, and was up all night with diarrhœa. By a very remarkable coincidence, a woman, aged 52, living two doors off on the same side of the street, was removed to the infirmary on September 25 with the most violent purging and vomiting. She died within three days, the cause of death on the death certificate being returned as "Simple enteritis."

The details of Case 2 are as follows: A girl, aged 2, living on the first floor, fell ill at the end of the first week in September, the approximate date of onset being arrived at from the fact that she was known to have been suffering from diarrhœa on the day when her mother was confined—namely, September 9. The child was subsequently notified to

TABLE VII.—MULTIPLE CASES OF DIARRHŒA IN HOUSES AND FAMILIES WHERE NO CASES OF FATAL DIARRHŒA OCCURRED.

House No.	Family surname	Age of patient	Date of onset	Remarks
1	B	1½	Sept. 10	Very severe, with vomiting
	A	25+	Sept. 23	
	A	2	Sept. 24	
	A	25+	Sept. 30	
2	A	2	Sept. 9	Contact between these two families was definitely proved
	B	16	Sept. 15	
	A	8	Sept. 16	
	B	2	Sept. 28	
	A	25+	Sept. 29	

me by the almoner of St. Mary's Hospital. She was looked after by a neighbour, occupying the ground floor in the same house, until her mother was able to return to her household duties after her confinement. On September 15 the neighbour's son, a boy aged 16, was seized with the most violent diarrhœa. So urgent were the calls to stool that he not only failed to reach the closet before his bowels were moved, but could not even leave his bed. On the same day, or the day following, the sister of the primary case, a girl aged 8, was attacked with diarrhœa. On September 28 her baby-brother, aged 2, developed diarrhœa, and the following day the mother of the primary case failed with the same disease in a severe form.

The evidence to be derived from the occurrence of these non-fatal attacks should perhaps be regarded as inadmissible, since they are not directly associated with any death certified by a medical practitioner as due to epidemic diarrhœa, and yet it is difficult to believe that a disease

which manifests itself by such violent symptoms does not account for large numbers of the infantile deaths which occur during the summer and autumn months.

CONCLUSIONS DERIVED FROM CASE HISTORIES.

The histories of the fatal and non-fatal cases which have now been given in detail might obviously be interpreted as suggesting that diarrhoea is a communicable disease, the infection of which is capable of being conveyed to healthy persons in the excrement of the sick. Since time will not permit the application of any other hypothesis to the facts set forth, another class of phenomena tending to show that diarrhoeal stools possess infectious properties will now be considered.

THE EFFECT OF WATER-CARRIAGE SYSTEMS OF SEWERAGE.

Longstaff (1891, p. 288) dismissed as untenable the theory that filthy privies have any direct bearing on the causation of diarrhoea, pointing out that privies are as common or commoner in the country than in large towns, and are for this reason unlikely to be the cause of a town disease. On the other hand, in Malta diarrhoea is not especially a town disease; and Newsholme (1899, p. 155) has shown that towns with the water-carriage system have, as a rule, much less diarrhoea than those retaining other methods of removal of excrement. In Ipswich a very remarkable decline in the prevalence of enteric fever has occurred in the past six years; and Pringle (1909, p. 415) has brought forward striking evidence to show that the decline has been due to the abolition of middens.

Nottingham, a town containing large numbers of pail-closets as compared with Leicester, a water-closet town, occupies an unfavourable position in regard both to its enteric-fever and diarrhoeal death-rates; and Boobyer (1908, pp. 51-63) attributes the steady diminution in the prevalence of diarrhoea and enteric fever in Leicester to the substitution of water carriage for dry removal.

In Ipswich the number of middens has been reduced from 8,000 in the year 1893 to 110 in 1908. Leicester, according to the figures given by Newsholme (1899, p. 207), contained 2,000 middens, 7,000 pail-closets, and 20,000 water-closets in 1896, and has now completed the conversion of the system of dry removal to a system of water carriage. Nottingham, and Wigan which is added for the purpose of the present inquiry, are pail-closet towns. London has been chosen as an example of a district

in which the water-carriage system has been established for a number of years. The diarrhoea and enteric-fever experience of these five towns during the past twenty years has been represented in twelve charts, which will be found on analysis to suggest the following conclusions:—

(1) In two towns where a decline in the prevalence of enteric fever has coincided with, or followed, the substitution of a water-carriage system of sewerage for dry removal, the diagrams show a closely corresponding fall in the death-rate from diarrhoea. The line denoting annual variations in the diarrhoeal death-rate in these districts shows that of

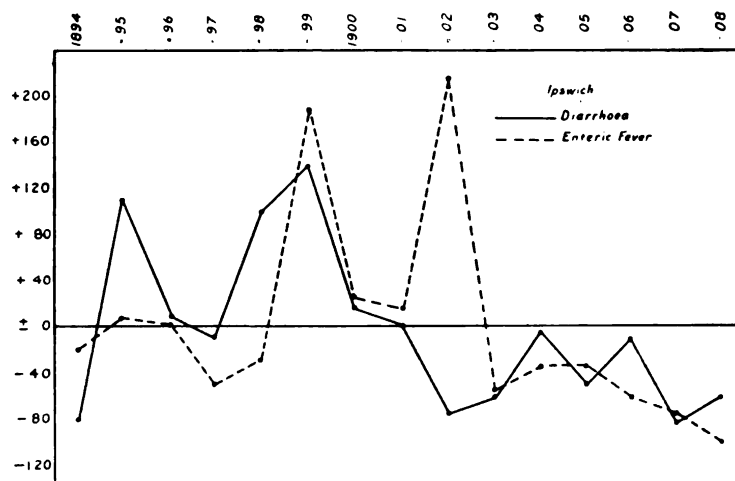


Fig. II. Percentage Variations from the Mean of Death-Rates from Diarrhoea and Enteric Fever in Ipswich in the years 1894-1908. Mean Death-Rate from Diarrhoea, 1894-08 = 1.05 per 1,000 living; from Enteric Fever = 0.15. The Death-Rates used are those given in the Annual Summaries of the Registrar-General.

late years the epidemic peaks have become less conspicuous, whilst epidemic peaks in the case of enteric fever have been practically obliterated. These points are illustrated in figs. 2, 3, and 4, which relate to Leicester and Ipswich, two towns where water-closets have been substituted for pails or middens during the past twenty years.

(2) Where no alteration has been made in the system of excrement removal, there is no decline in the death-rate from diarrhoea—as, for instance, in Wigan, London, and Nottingham—whilst enteric fever either remains stationary, as in Wigan, or, if it diminishes, the experience of London and Nottingham suggests that the decline is of a different character in midden and water-closet towns respectively. From the

beginning to the end of the enteric-fever curve for Nottingham epidemic peaks recur; in the chart for London epidemic peaks are less conspicuous throughout, and are practically obliterated in the last eight annual periods. These points are illustrated in figs. 5, 6, 7, and 8, which relate to Wigan and Nottingham, two towns still retaining large

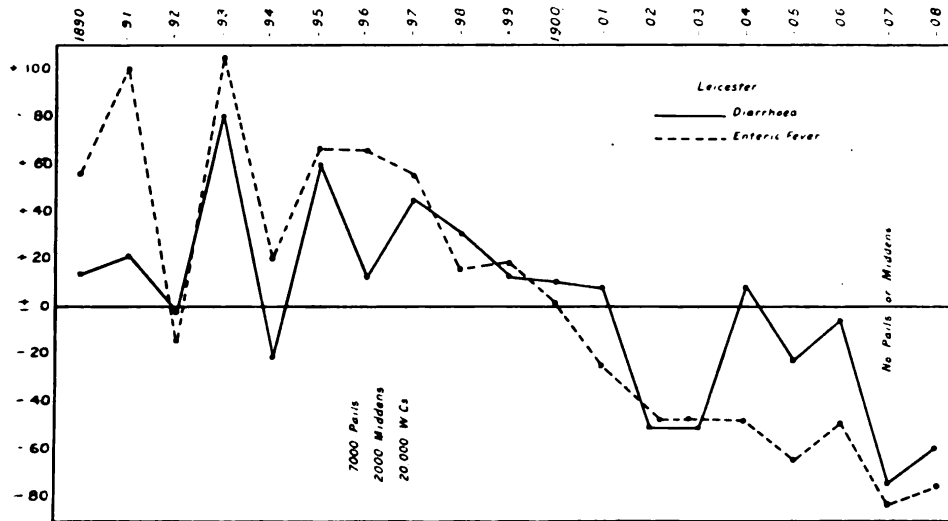


Fig. III. Percentage Variations from the Mean of Death-Rates from Diarrhoea and Enteric Fever in Leicester in the years 1890-1908. The Mean Death-Rate from Diarrhoea for the period 1890-1908 was 1.21 per 1,000 living. The Mean Death-Rate from Enteric Fever for the same period was 0.12 per 1,000 living.

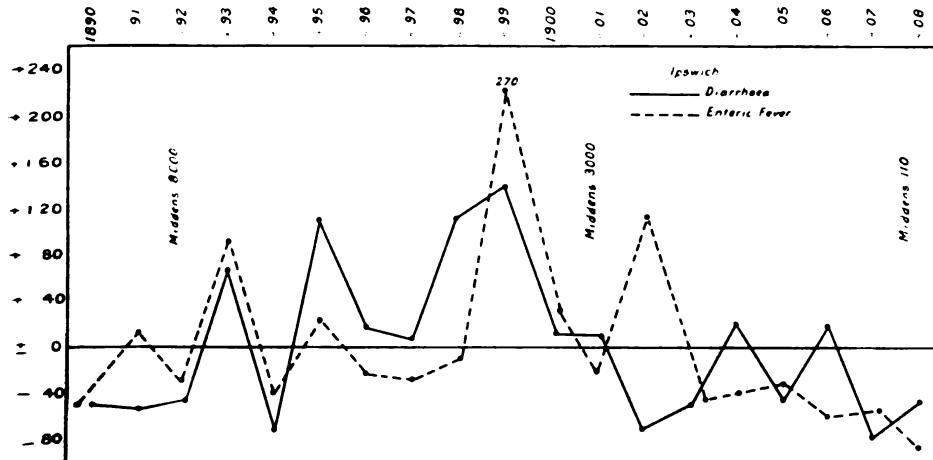


Fig. IV. Variations in Death-Rates from Diarrhoea and Enteric Fever Case-Rates in Ipswich in the years 1890-1908. The Mean Death-Rate from Diarrhoea was 0.95. The Mean Case-Rate for Enteric Fever was 1.18 per 1,000 living. Dr. Pringle has included deaths from Gastro-enteritis and Enteritis in calculating the Death-Rates from Diarrhoea on which the table is based.

numbers of pail-closets, and to London, where the water-carriage system has been established for many years.

(3) Fig. 6 for London, compared with fig. 7 for Nottingham, also shows a smoother curve for diarrhoea with smaller variations from the mean death-rate, in marked contrast to the high peaks and deep valleys which represent the fluctuating death-rate from diarrhoea in the midden

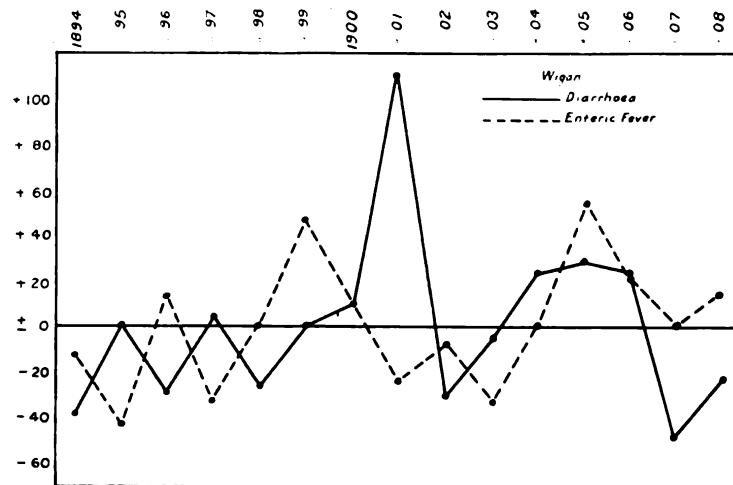


Fig. V. Variations of the Death-Rates from Diarrhoea and Enteric Fever in Wigan during the years 1894-1908. The Death-Rates are taken from the annual summaries of the Registrar-General, the Mean-Rate for Diarrhoea in the years 1894-1908 being 1.30 per 1,000 living, and the Mean-Rate for Enteric Fever being 0.28

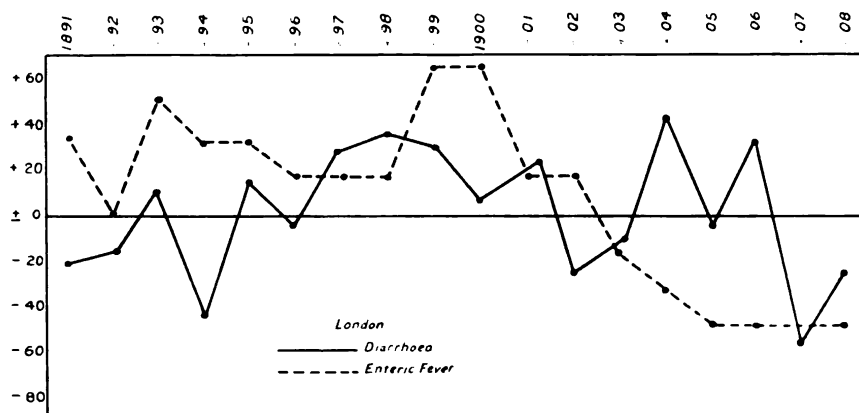


Fig. VI. Variations of the Death-Rates from Diarrhoea and the Case-Rates from Enteric Fever in the years 1891-1908 in London. The Death-Rates from Diarrhoea are taken from the Registrar-General's Report for 1908, the Mean-Rate 1891-1908 being 0.72 per 1,000 living. The Case-Rates for Enteric Fever are taken from the Annual Reports of the Medical Officer of Health for the County of London, the Mean-Rate being 0.6 per 1,000 living for the years 1891-1908

town. Fig. 9 illustrates a similar contrast in the diarrhoeal curves for Leicester and Nottingham.

(4) Fig. 10 shows that, without any alteration in the sewerage system, the number of cases of enteric fever in London have fallen to almost the same extent as in Ipswich, but it also illustrates in a graphic manner how the curve in Ipswich, with the adoption of water-closets, has taken on a striking resemblance, in the absence of peaks and valleys, to the

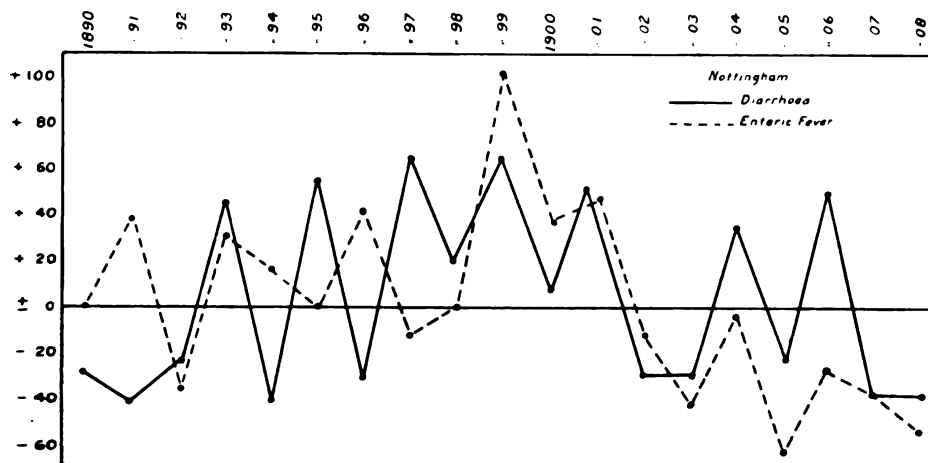


Fig. VII. Variations in the Death-Rates from Diarrhoea and Enteric Fever in Nottingham during the period 1890-1908. The Mean Death-Rate from Diarrhoea was 1.04 per 1,000 living. The Mean Death-Rate from Enteric Fever was 0.24.

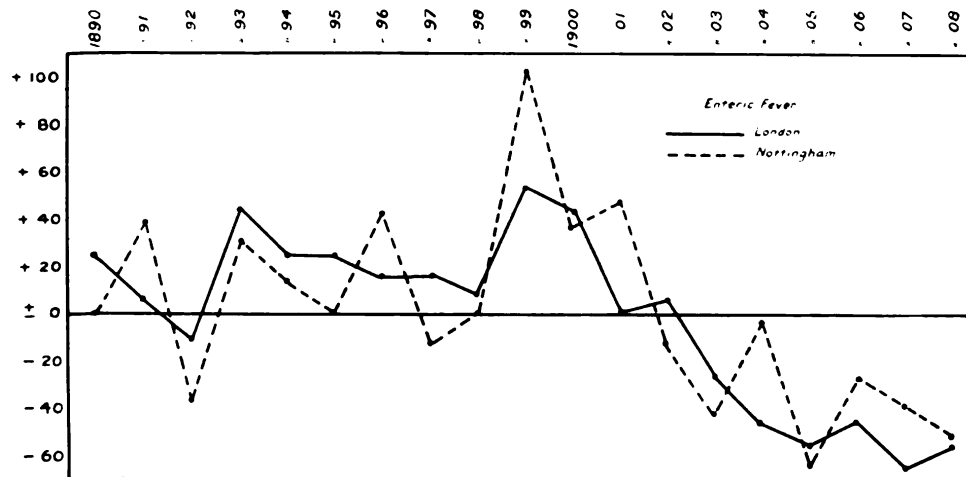


Fig. VIII. Variations in the Death-Rates from Enteric Fever in the years 1890-1908 in London and Nottingham. The Mean-Rate was 0.11 in London and 0.24 in Nottingham.

curve of London, a water-closet town. If the altered form of the Ipswich curve only extends over the last six annual periods, it must be remembered that when the last peak rose high above the mean in 1902, some 3,000 midden privies were still in use. Fig. 11 illustrates the very marked contrast between the rates at which enteric fever has declined in Leicester and Nottingham respectively, and further shows in a strik-

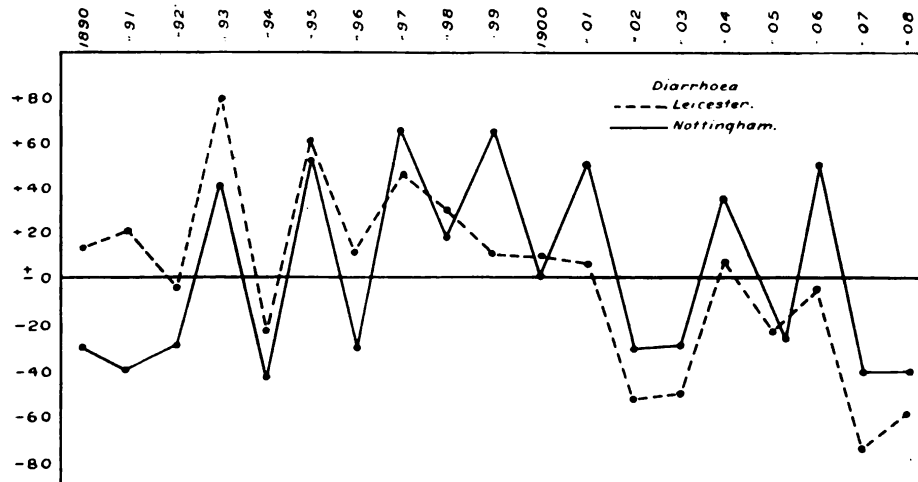


Fig. IX. Variations of Death-Rates from Diarrhoea in Leicester and Nottingham in 1890-1908. The Mean Death Rate from Diarrhoea per 1,000 living, in the period 1890-1908, was 1.21 in Leicester and 1.04 in Nottingham.

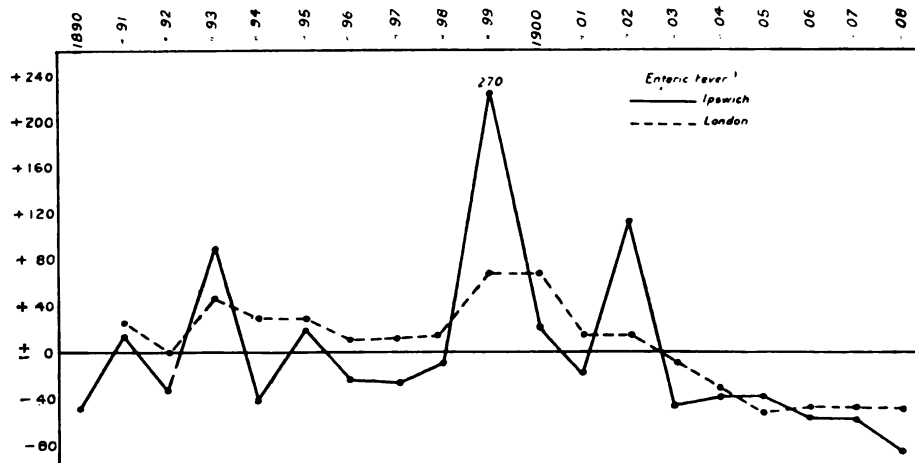


Fig. X. Variations in Enteric Fever Case-Rates in 1890-1908 in London and Ipswich. The Mean Case-Rate in London for the period 1891-1908 was 0.6 per 1,000 living. The Mean-Rate for Ipswich in the period 1890-1908 was 1.18 per 1,000 living.

ing manner the difference between the peaked curve of a privy town and the undulating line which has come to represent the annual death-rate from enteric in Leicester since middens were abolished.

Fig. 12 shows the contrast between the diminishing death-rate from diarrhoea in Ipswich and the stationary death-rate in Wigan. The epidemic peaks of Nottingham are not reproduced in the chart for

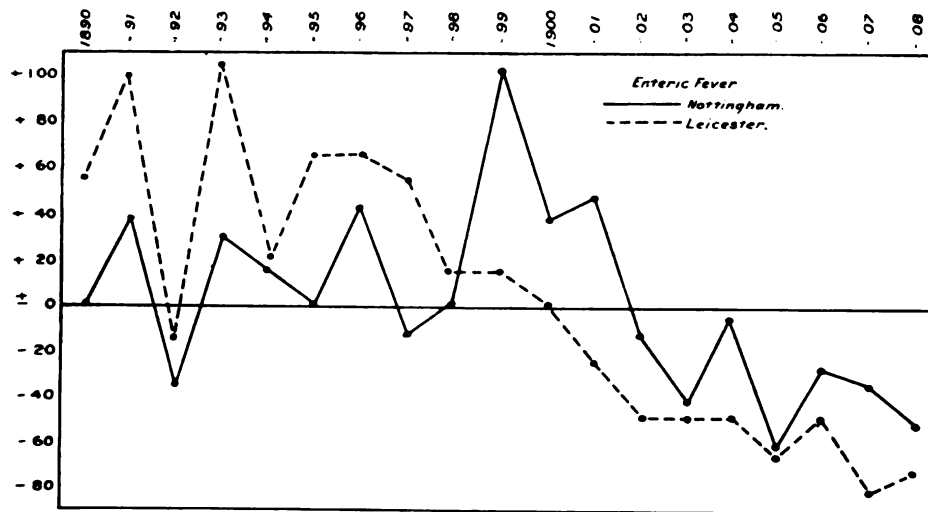


Fig. XI. Variations in the Death-Rates from Enteric Fever in 1890-1908 in Nottingham and Leicester. The Mean Death-Rates from Enteric Fever were 0.24 in Nottingham and 0.12 in Leicester.

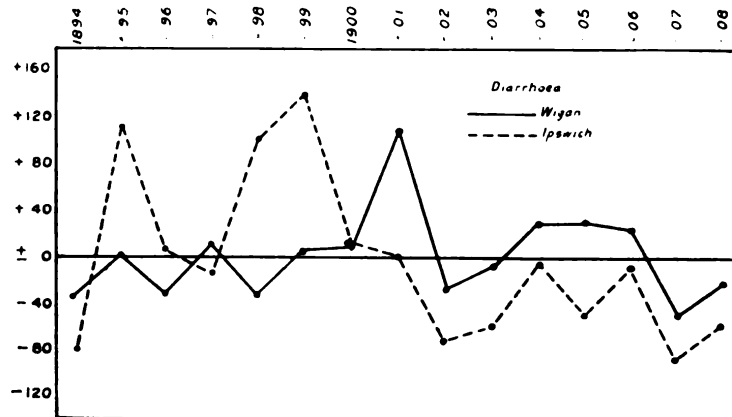


Fig. XII. Variations in Death-Rates from Diarrhoea in Wigan and Ipswich, 1894-1908. The Mean Death-Rate from Diarrhoea in Wigan was 1.80 per 1,000 living. The Mean-Rate for Ipswich was 1.08.

Wigan, a possible explanation being that the death-rate from diarrhoea is maintained at so high a level in normal years as to be incapable of showing a well-marked rise under those weather conditions which beget epidemic outbreaks of diarrhoea in other towns.

CONCLUSIONS FROM CHARTS.

In summarizing the points illustrated in these diagrams it may be said that the prevalence of two intestinal diseases has declined in a very remarkable manner in two towns where the practice of exposing human excrement in pails and middens has recently been abolished; and, further, that both diseases are due to bacteria which are known in the one case and assumed in the other to reside in the faecal excrement of infected persons. The interpretation of the decline in enteric fever is beset with difficulties, since the prevalence of this disease is diminishing not only throughout England but in many other European countries; and the decrease, as we have seen, may occur indifferently in midden or water-closet towns where the sewerage system has remained unaltered for many years. Since no data have been collected with a view to determining the relative importance of local and general causes, the case of enteric fever must be dismissed with the plea that the universal operation of some essential unknown factor cannot prevent the added influence of secondary local causes from taking effect, neither can the experience of London be held to constitute a direct negation of the theory that the abolition of privy middens has been the direct cause of the greater part of the diminution in the enteric-fever death-rate which has been recorded in Ipswich and Leicester.

With diarrhoea there is not the same difficulty. In the Maltese villages, in Wigan, Nottingham, and London, the disease has not become less prevalent, neither has any alteration in the sewerage systems of the districts been made within recent years. In Leicester, Ipswich, and the Maltese towns the fall in the diarrhoea death-rate has coincided with the introduction of water-closets in place of privies.

It remains to be considered whether the case for diarrhoea as a communicable disease has been strengthened or weakened by reference to enteric fever, since we cannot reverse the problem and argue from data derived from the behaviour of diarrhoea, a doubtful entity, in support of typhoid fever, a specific disease, which is known under certain circumstances to be contagious. In this connexion it is important to

note that the cases of diarrhoea in older children and adults which occurred in Kensington reproduce in miniature the age-incidence of the disease reported by Ballard (1887-8, pp. 23, 26). Of 4,500 cases of diarrhoea recorded in Islington, no fewer than 1,700—that is to say, more than one-third—occurred in persons over 10 years of age. Among 44,000 cases tabulated by Dr. Ballard in Leicester, 75 per cent., or more than 33,000, occurred in persons over the age of 5 years. If only a small proportion of these persons suffered from an infectious form of diarrhoea, the amount of infectious excrement passed into privies would have exceeded the amount passed by persons suffering from recognizable enteric fever. It follows, therefore, that unless the abolition of privies can be shown to produce the same reduction in the prevalence of diarrhoea as it appears to produce in typhoid fever, the claim that diarrhoea is capable of being communicated by the same channels as the typhoid fever of military camps would become so weak as to be almost untenable. A single test case—namely, that of Ipswich—has been taken at random, and has shown the same correspondence in the decline of the two diseases as was noted by Dr. Boobyer in Leicester.

If the analogy with enteric fever be abandoned, the experience of Malta, Wigan, London, Nottingham, Ipswich, and Leicester constitutes a strong argument in favour of the infectious nature of diarrhoeal stools.

Further evidence pointing in the same direction was obtained by Longstaff (1891, p. 302) from the Medical Officers of Health of Liverpool, Chester, and Worcester, who attributed the decline in the diarrhoeal death-rates in their districts to the “substitution of water-closets for privies”; and finally Dr. Boobyer (p. 63) has laid stress on the “conversion or partial conversion of dry closets” in Manchester, Salford, and Birmingham as an important factor in bringing about the reduction in the diarrhoeal mortality which has occurred in these towns.

CONCLUSIONS.

Two formidable difficulties have to be faced by every one who sets out to consider the epidemiology of diarrhoea. The first, which is common to all apparently communicable diseases, is the difficulty of determining the relative parts which have been played by the three factors, common environment, contact, and coincidence, in the production of any given group of phenomena observed. The second arises from

our inability to distinguish from one another the disease we are considering and the disorders which it simulates. The following conclusions are not therefore set forth without a full sense of the obscurity surrounding the evidence on which they are based.

If diarrhœa is communicable, it is important to define the paths by which the infection is conveyed. In a disease so highly contagious as measles the conclusion cannot be avoided that the healthy may acquire infection from the sick through mere proximity in the same room. On the other hand, the rarity with which case-to-case infection occurs in typhoid fever renders it almost certain that healthy persons can only acquire the disease from others in their neighbourhood by the ingestion of food which has come into contact with infected urine or fæces. Infection by these means in privy towns is readily conceivable, but in water-closet towns cannot account for large numbers of cases.

There is evidence in this paper amounting to proof that certain fatal forms of summer diarrhœa are communicable, and Dr. Low has brought forward conclusive evidence to the same effect. Nevertheless communicability is by no means a conspicuous feature of epidemic diarrhœa in every case. Thus in nineteen out of thirty-five tenement houses in Kensington where deaths were registered as due to diarrhœa, no other cases occurred. Again in twenty-five out of thirty-five fatal cases of diarrhœa, no source of infection was found in the families occupying the houses where those patients died. And, lastly, in twenty-two families containing young children fatal diarrhœa occurred, and yet in these families forty-four parents and fifty-eight children were intimately exposed to infection without falling ill. In five hospitals there is no evidence of the spread of diarrhœa from patient to patient, and the sum of the evidence suggests that diarrhœa is not more infectious than typhoid fever, and is not conveyed except by the same channels. There is, however, this difference between the two diseases—namely, that typhoid fever spares young children, whereas diarrhœa selects them for its victims. Obviously young children suffering from diarrhœa cannot avoid contaminating their surroundings with fæcal excrement to a much greater extent than adult persons, and for this reason in districts with a water-carriage system of sewerage diarrhœa will be more likely to spread from the sick to the healthy than typhoid fever.

Abstract considerations such as these can, with the aid of the house fly, be made to fit the theory that every case of summer diarrhœa is derived from the fæcal excrement of a previous case; but practical experience of single attacks in persons living far removed from other

patients and of groups, the members of which show some proximity on a map, though actually separated from one another by considerable distances of space and time, suggests that the manifestations of diarrhoea observed in water-closet towns neither disprove the existence of impersonal sources of infection in many fatal cases nor demand the rule of universal human origin which they can be forced to support.

In privy towns, whilst heterogeneous filth and sewage-polluted soil may exert the influence attributed to them by Ballard, the phenomena observed can, without any undue strain, be fully explained on the assumption that the effective cause of fatal summer diarrhoea is conveyed to the healthy in the freshly-passed excrement of the sick, and is not acquired from any other source.

In conclusion, I wish to take the opportunity of expressing my sincere gratitude for their valuable assistance to Professor W. Osler, of Oxford University, Dr. Pringle, of Ipswich, Dr. S. T. Irwin, of Belfast, and especially to the Resident Medical Officers of a number of children's hospitals, who have furnished me with information of the greatest value.

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DISCUSSION.

Sir SHIRLEY MURPHY said he thought that, for fairness of statement and of inference, the Section had never had a paper which commended itself more to him. One of the observations he would make upon the paper was that it showed the extreme difficulty of distinguishing between attacks which might be due to a common cause and those which might be suggestive of infection from case to case. Taking the outbreaks which occurred in a house or group of houses where all the members were living in the same conditions, it was difficult to find facts which conclusively demonstrate infection from case to case. The facts were consistent with the hypothesis that the populations in question were all more or less exposed to the same conditions, and that the resultant cases or groups of cases, whether in houses or streets, might be due to a common cause. He thought the information which came from the hospitals, where, of course, the patients were not living under the conditions of their homes, was more free from this objection. One of the points which required consideration in studying diarrhœa, especially when comparing one town with another, was the social condition of the people, because that social condition governed the liability to attack. He had had prepared a statement of the incidence of attack upon groups of London districts, the districts being grouped according to social conditions, the criterion being the proportion of the child population attending the elementary schools. In the last four years, taking the deaths from diarrhœa in proportion to the births, it was found that in groups of districts where less than 61 per cent. of the children attended elementary schools, the mortality was a trifle over 20 per thousand; from 61 per cent. to 66 per cent., the mortality was about 22 per thousand; from 66 per cent. to 71 per cent., 24 per thousand; from 71 per cent. to 76 per cent., 28 per thousand; above 76 per cent., 30 per thousand. Therefore the mortality from diarrhœa had absolutely followed the social condition of the people. In comparing such towns as Nottingham and Leicester, it was necessary to know the social condition of the people. It was only those who lived in the towns who were in a position to form a proper estimate as to the influence of this condition. When one heard that one town was improving in health and another was not, one wondered whether there had been any alteration in the social conditions of the people which, apart from the replacement of privies by water-closets, might have affected the result.

Dr. RALPH VINCENT said that he had listened to Dr. Sandilands' paper with great interest, and desired to express his appreciation of the fairness and impartiality with which Dr. Sandilands approached the study of a difficult subject. The study of the circumstances of the communicability of disease, of "multiple invasion," and of allied questions, was beset with fallacies that

tempted the unwary, and he could not but congratulate the reader on the scientific method which was the distinguishing feature of his paper. His (Dr. Vincent's) paper on the "Ætiology of Zymotic Enteritis" had been published so recently that he would content himself with drawing attention to certain specific points. In the first place he stated, without any qualification, that zymotic enteritis is not an infectious or contagious disease. But, indeed, he went much further than this. The disease cannot be conveyed by means of infection. By this he meant not merely that the disease, as it is commonly found, does not behave as an infectious disease, but that the processes of the disease are essentially not infective in character; for the organisms associated with the production of the disease cannot live in the tissues of the living body. In enteric fever, for instance, the organisms are found in the blood, in the liver, in the spleen, and in other organs. In zymotic enteritis the organisms are never found in the tissues—not even in the tissues of the alimentary canal, but only in its contents. This is a fundamental distinction; and when he stated that the disease is not infectious, he meant a great deal more than simply that the disease is not a "catching" disease. He ventured to lay some stress upon this, for it was clear to him, from numerous communications which he had received, that the essential point in his account of the disease had not been at all clearly understood. A remarkable fact which had lately come prominently to his knowledge is the confusion which exists as to what constitutes an infectious disease. A well-known Medical Officer of Health, for example, had written to him: "I cannot agree with you that the fact that the disease is not spread in the infants' hospital is a proof that the disease is not infectious," and the view thus expressed was by no means singular to himself. He could only say that he was at a loss to imagine what were the writer's criteria of infectious disease. For himself he quite accepted the ordinary characteristics as laid down by Ballard, and he certainly agreed with Dr. Sandilands when he said: "To these characteristics should certainly be added the occurrence of any given disease, outside its normal environment, among the inmates of hospitals to which infected persons have been removed." He had a vivid recollection of the attacks levelled at them when the infants' hospital was first established, and the following extract from the medical report for the year 1904 was, he thought, of sufficient interest to quote, for it afforded encouraging evidence of the progress that had been made since then:—

"At the time of the establishment of the hospital, and ever since, we have been met with one criticism on all hands—namely, that it was not possible to keep a number of even healthy babies in a ward with satisfactory results. The experiment, it was said, had been tried repeatedly, and had always failed. So great was the mortality in one instance mentioned that 96 out of 100 died. The reason for these failures was probably always the same—namely, impure milk or inadequate methods of substitute feeding. The summer of last year [1904] was extremely hot, and the infantile mortality throughout the country was much higher than in previous years. Yet, in the hospital, not a single case of 'epidemic diarrhoea' (zymotic enteritis) developed, despite the fact that on one occasion there were six cases in the wards, brought to the hospital, suffering from the disease."

It must be borne in mind that in the hospital they took no precautions such as are taken in the case of infectious disease. If it were an infectious disease, then, in the late summer, the whole hospital should be down with it, patients and nurses alike, for there was continual contact. In at least one children's hospital the disease is regarded as an infectious disease. Strict precautions are consequently taken. The infants suffering from the disease are isolated in a special ward. The nurses feeding the infants do not change them, and the whole ritual of antiseptic precautions is systematically carried out. And with what result? Case-to-case "infection" is alleged to occur, not only among the patients, but even among the nursing staff; while at the infants' hospital where all such precautions are conspicuous by their absence such a thing is unknown. Surely this was a strange account of an infectious disease. It is, however, when one is faced with the clinical facts of the disease that the infective theory breaks down altogether. No doubt, in many instances, it might be easy to demonstrate cases which could be explained either by common environment or by case-to-case infection. But it was equally certain that large numbers of cases arose in which no sort of case-to-case infection could be traced. How were these to be explained? The true explanation must provide for all cases. How was it that the breast-fed infant escaped? The difficulty here was so great that men of judgment and experience had been driven to adopt arguments which they could not have used except under the obsession of the specific infective theory. One Medical Officer of Health attributed the disease to the dirty "comforters" sucked by infants. Was there a special brand of clean "comforters"—the monopoly of breast-fed infants? Another Medical Officer of Health emphatically pointed out the immunity of breast-fed babies; but he was so pressed to harmonize this with his conception of the disease that he suggested that the immunity of these infants was probably due to the fact that breast-fed babies do not crawl about the floor quite so much as do the bottle-fed babies. Let it be acknowledged that these remarks were a little dragged out of the context. He cited them in no controversial spirit, but as evidence of the extremities to which the infective theory of zymotic enteritis led us. For his own part he could only say that, in the last ten years, he had been engaged in the observation and treatment of large numbers of infants. He began, of course, with the current explanation of "epidemic diarrhœa." He abandoned it, simply because he could not go on with it; it would not work. It could be dragged in, by hook or by crook, to explain a certain number of cases; in very many cases it would not fit in with the facts at all. In order to study disease it was essential to study health. What are the conditions under which the healthy infant lives and thrives, whether breast-fed or bottle-fed, and which render it immune from zymotic enteritis? It is useless to limit the inquiry to those attacked, and then demonstrate the part played by temperature, by dust, by flies, by multiple invasion, &c. For flies alight upon the lips of the breast-fed baby; the same kind of dust enters

the mouth of the babies, naturally fed and artificially fed. The essential feature of the account of the disease which he had formulated was that it did explain the facts, not only in regard to the infants who were attacked, but also in regard to those who were not attacked. Was there a single Medical Officer of Health of judgment and experience who could say the same thing of the theory that zymotic enteritis was an "acute infective disease"? He had been greatly impressed by one fact in reference to the numerous communications which he had received from medical men in various parts of the country relating to his paper. Dried milk, sterilized milk, &c., &c., were all referred to, but he had not received a single letter drawing his attention to cases of zymotic enteritis occurring in infants fed on raw milk. Fresh, uncooked milk appeared to be almost unknown as an article of diet among the infants of the poorer classes who were not breast-fed. That appeared to him to be a very serious matter indeed. The boiling of milk, as carried out by the mothers, afforded no protection from the disease; on the contrary, it was responsible for some of the worst and most fatal cases.

Dr. SIDNEY DAVIES desired to join in Sir Shirley Murphy's praise of the paper. The author had approached the subject from a cautious and scientific standpoint. It was interesting to hear Dr. Vincent absolutely opposing all that most of those present had accepted as true for many years, certainly since Ballard's contributions. Dr. Vincent asked why breast-fed infants did not get diarrhoea. But surely all recognized that the infection was passed on mainly indirectly—through the food, and especially through the milk. If that was so, obviously breast-fed infants would escape it. Apparently, Dr. Vincent doubted that infants who took raw milk ever had diarrhoea. He assumed that he would call milk raw if the heating had not been beyond body temperature. [Dr. VINCENT: Yes.] Surely an inquiry twenty years ago would show that most of the infants who had diarrhoea had milk raw in that sense. Twenty to forty years ago it was not the custom of the poor to cook milk before giving it to children; it was warmed only for greater palatability. He asked why Dr. Sandilands suggested (p. 110) that "the evidence to be derived from the occurrence of these non-fatal attacks should perhaps be regarded as inadmissible." Surely the evidence was very striking, and it was not necessary for a case to be fatal for it to be one of specific diarrhoea. Admittedly the great difficulty was what was to be regarded as such. It was in that respect that Dr. Vincent took a stand in which he (the speaker) could not follow him. Were there any infectious diseases in which mild cases did not occur? He was not aware of any. Still, it was difficult to say exactly what was a case of zymotic enteritis and what was not. A point in support of its communicability was the way in which, when once well started, it persisted. There was the simple fact that more cases occurred in September than in July. He supposed everybody regarded the temperature as one of the important

factors in the causation of the disease; but from Dr. Vincent's standpoint he would have expected much less diarrhœa in September than in July; the temperature reached its highest average about July 12. If the temperature simply acted directly in causing the increase of the non-specific bacteria and the food poison in the milk, surely there was nothing to account for the fact that the disease kept up its high level in September and October, which it was known to do. That might be explicable in more than one way: (1) There might be some ground influence affecting the germs there. A more likely explanation was (2) that infectivity having been started the disease was conveyed from case to case, even though the temperature had gone down. The argument seemed to be supported from the experience at Woolwich. That was the only part of London in which there had been general paid voluntary notification of zymotic enteritis during the past five years. He knew he was liable to the criticism which Sir Shirley Murphy made, but he had carefully considered the point. In the four years 1901-4 Woolwich had a higher diarrhœa death-rate than London—viz., 2'30 as compared with 2'27. In the succeeding four years the mortality from diarrhœa in Woolwich came down to 1'37, while the London mortality fell to 1'78 only. The figures for 1909 would still further emphasize the comparison. He attributed the difference largely to the fact that, having notification, measures were taken to prevent spread from case to case in houses, and by flies in the immediate neighbourhood; though no doubt a large part of the reduction was due to the direct effect of the advice of the health visitor as to the treatment of the patient diminishing the fatality. The precautions advocated were on the basis of the disease spreading like enteric fever. There had been no upward trend in the social condition of the people of Woolwich which would account for the improvement; in fact, recently, the population had been decidedly poorer.

Mr. M. GREENWOOD, Jun., asked what was the average size of family in the Kensington series. In 22 of the families there were 102 persons, and if this were a fair sample one could take 5 to a family as a rough approximation. This would give 64 cases among 92 by 5 persons. Supposing the cases to occur at random among this population, one would expect, on the average, to find about 13 families containing 2 or more affected members. Actually there were 12. *Prima facie*, this did not suggest that the mere number of multiple infections in the Kensington series was evidence in favour of infectiousness. The assumption of an equal incidence upon all members of the families might not, however, be justifiable.

The PRESIDENT (Dr. Niven) desired to congratulate Dr. Sandilands on the admirable manner in which he had set forth the facts. The author was kind enough to make a complimentary speech about his (Dr. Niven's) annual reports, but after the facts which Dr. Sandilands had set forth so fully, he must, to a large extent, disclaim what that gentleman said about the presentment of

the facts. He (the speaker) believed that such things occurred in hospital oftener than Dr. Sandilands had been able to show ; though he did not like to admit it was any necessary part of a demonstration of that kind that those occurrences should take place in hospital. Surely no one doubted that tuberculosis was a communicable disease, yet it was constantly maintained that the disease did not spread in hospitals in this country ; so it was going far beyond the necessities of the case to claim that the disease must be spread in hospital. In poor districts it was common to see enteric fever spreading through family after family, many members of such families being affected. On the other hand, although he thought the aggregate of the cases which Dr. Sandilands had produced, and of those which he (the speaker) had collected, showed that the disease was in some way communicated from one person to another in that series, he was not sure that it was always directly communicated. Dr. Sandilands mentioned odd cases in which the disease was on the other side of the street, or in a part of the house where there was no evident contact, and so one must take into account the evident possibility that some intermediate agent was present ; perhaps flies, or some other animal agency, carrying the infection from one part of the premises to another ; and, if that took place outside the family, it might equally occur within it. So he thought one must take the histories given as largely the histories of infection. In reference to Dr. Vincent's remarks concerning raw milk and boiled milk, he (Dr. Niven) had collected a quantity of material in reference to the foods which the children had had ; and he had tried to look at the other side of the question of transmission. The boiling of the milk had been a definite question in the inquiry forms, from the answers to which it was clear that many of the children were given raw milk. Some were said to have habitually had boiled milk. So rawness of milk was no protection against the communication of the disease. He agreed there might be high temperatures and yet no diarrhoea ; something required to be superadded to the temperature to bring about the diarrhoea. In well-to-do towns in the south of England there was nothing like the same amount of diarrhoea which was found in Lancashire towns, and in his opinion part of the something else was the predisposing weakness in the child, and part flies, which transmitted the infection. With regard to breast-fed infants, if it was once admitted that the disease was carried by flies, it was easy to see that flies clustered about the mouth and nose of infants to get moisture, and in that way they might infect breast-fed infants ; but probably the commonest mode of infection was by the food. He did not propose to discuss the paper further, but he had been much struck by the author's clearness in connecting the reduction of the disease with the abolition of middens. He appreciated the point made by Sir Shirley Murphy, but he thought that point had been in a measure met, and that there could not have been a change socially in such towns as Leicester, which would remove the force of the demonstration which Dr. Sandilands had given.

Dr. SANDILANDS, in reply, said he had been very much struck by the connexion between diarrhœa and poverty in Kensington. It occurred to him that this connexion might be explained firstly by aggregation, secondly by the preponderance of young children amongst the poor, and thirdly by the greater tendency for poor children to contaminate their surroundings with excrement. These children habitually micturated in the streets up to the age of 8 or 9 years, and nothing took the place of the napkins which were discarded before the ninth month until the age of 3 to 3½ years. Playing uncared for in the streets, a young child with looseness of the bowels would inevitably defæcate in any passage, yard, or footway where it happened to be at the time. Sir Shirley Murphy had pointed out the fallacy of comparing one district with another in the absence of accurate information as to the relative social conditions prevailing in the two districts compared. Obviously the mere comparison of the total mortality from diarrhœa in two towns would be most fallacious; but all reference to total mortalities had been purposely avoided. Two towns had been taken where the system of excrement removal was known to have been revolutionized in the last twenty years. In both the diarrhœal death-rate had fallen. Had this fall been shared by other towns (where the sewerage system had remained unaltered) as a result of a general progress in sanitation? The experience of Wigan, Lincoln, and Nottingham showed that it had not. He agreed with Dr. Vincent that it was the custom of poor people to boil their milk; he had found this practice to be almost universal in Finsbury, where he had visited very large numbers of poor people. Dr. Vincent appeared to attribute diarrhœa to putrefactive changes occurring in milk either before or after it had been consumed by the infant attacked. When these changes occurred after the milk had been consumed, they were due to certain abnormal conditions of the intestinal secretions which resulted from chronic dyspepsia. A fault in this theory of Dr. Vincent's was that it did not afford any sufficient explanation of the diarrhœa of adults. Dr. Davies had asked why he (the speaker) did not accept his own non-fatal cases as being undoubted cases of epidemic diarrhœa. The reason was that he had been obliged to rely in these cases upon histories furnished by the mothers of the families attacked: in the fatal cases there was the added security that death had been certified by a medical practitioner as due to diarrhœa. He, of course, was prepared to accept trivial attacks of diarrhœa as possible manifestations of zymotic enteritis provided it was conceded that a certain diagnosis in such cases could not possibly be made in our present state of knowledge. He had included mild cases among instances of multiple invasion, but the histories where direct infection had been suggested only related to severe diarrhœa often accompanied by vomiting. He was strongly of the opinion that the milk became contaminated in the home, and that, for this reason, boiling the milk when it was received was a useless precaution. Dr. Butler had suggested boiling each feed immediately before it was given, but this meant that each feed must also

be cooled again, and it was doubtful if the busy mother would have time to carry out Dr. Butler's suggestion, which was eminently sound, except from the domestic point of view. The President had argued that, although typhoid fever rarely spread from case to case in hospital, it nevertheless was readily communicated by contagion in the homes of the poor. He had gone on to suggest that the contagious properties of diarrhoea might vary in the same way under the influence of environment. In answer to the President's argument, it might be contended that the autumnal waves of typhoid fever, at any rate in water-closet towns, were very far from having been proved to be due to case-to-case infection, and that only those facts which were established in the epidemiology of typhoid fever could be safely used as a guide to the probable course of events in diarrhoea. Dr. Davies, in criticising Dr. Vincent's theories, pointed out the want of correspondence in time between the onset of hot weather and the occurrence of high diarrhoeal mortality, and that was a very valuable criticism, because at the time when milk began to be most liable to putrefaction, and therefore, according to Dr. Vincent's theory, most dangerous, the diarrhoeal mortality was insignificant; but when the temperature had fallen, and milk had a tenfold better chance of keeping in the first cold nights of September, the diarrhoeal mortality still continued to be excessive.

Dr. J. T. C. NASH wrote: I adhere to the opinion I have frequently expressed in print—that there is no one organism which can be claimed or regarded as “the causative agent of diarrhoea.” In any particular outbreak of diarrhoea there may be, no doubt, a particular organism, or perhaps a symbiotic group of organisms, which may be looked upon as specific for that particular outbreak; but in another diarrhoea outbreak in a different locality quite a different organism, or organisms, may be playing the chief causative part. Further, I adhere to the opinion that the relative numbers of organisms with putrefactive properties present in a food supply (particularly milk) are of extreme importance in rendering those foods capable or incapable of producing diarrhoea. Many milk supplies improperly managed contain such putrefactive organisms which in cool weather, however, do not multiply sufficiently to be able to cause a sufficient amount of chemical change in the milk to cause diarrhoea, while in warm weather the multiplication of similar organisms, and the consequent formation in excess of diarrhoea-causing toxins, is so rapidly effected that milk obtained under the same conditions as may have proved harmless in winter may now, as a result of the enormous numerical increase of these organisms in warm weather, excite diarrhoea. I certainly hold that such diarrhoea-causing organisms are, during the fly season (the *Musca domestica* season), largely carried to milk by flies—not only on the legs, wings, and bodies of these obnoxious insects, but to a very important degree through the intestines. It is a common observation that a frightened or drowning animal evacuates its intestinal contents, and I have no doubt that this occurs in the case of flies drowned in milk. Indeed, I have often observed that the ovipositor of a drowned fly is

completely obtruded. The crowding of houses in a given area possibly is an important factor in the spread of diarrhœa, and here again this may be explained by the increased opportunities given to a larger number of flies to peregrinate and carry larger and more numerous quantities of infective material from one house to another. The infection of nurses is easily understood and could easily be effected and accounted for in at least two ways: (1) By direct infection of the nurse's hands from soiled sheets or napkins and imperfect cleansing of the hands before putting them to the mouth unintentionally, or in eating bread, &c.; (2) the indirect contamination of the nurse's food through the agencies of flies. (Flies swarm in great numbers round the beds of children suffering from diarrhœa.) Where proper precautions are taken neither nurses nor other patients in hospital need become infected, as is illustrated by some of the examples given by Dr. Sandilands. With regard to multiple cases in houses, it must not be overlooked that, although the subsequent cases may be secondary to the first, they may, on the other hand, be altogether independent of it; and be instances of fresh pollution of food through the agency of flies, quite apart from the first case. The probabilities, however, of flies now carrying more specific contamination than before the occurrence of the first case are obviously greater, and without doubt a considerable proportion of the later cases are really secondary. Unquestionably, midden-privy towns, as a rule, suffer more from diarrhœa than water-carriage towns. I do not propose to discuss this further than to refer to my remarks on this subject in a paper on "The Prevention of Epidemic (or Summer) Diarrhœa."¹ The epidemiology of diarrhœa is a very large subject, and investigations like those made by Dr. Sandilands and embodied in his interesting paper will add to or confirm our existing knowledge and working hypotheses. Such published records as have been made by Dr. Niven, and now by Dr. Sandilands, not only give us useful information, but suggest further lines of inquiry which may be prosecuted in the future.

¹ *Practitioner*, 1906, lxxvi, p. 708.

Epidemiological Section.

April 7, 1910.

Dr. JAMES NIVEN, President of the Section, in the Chair.

Summer Diarrhœa and Enteric Fever.

By JAMES NIVEN, M.B.

THIS paper is of the nature of an intensive study. For many years I have published in the annual reports on the health of Manchester and elsewhere, going back so far as 1883, facts and interpretations of facts in relation to enteric fever, and more recently in regard to diarrhœa. It is proposed to lay some of these before you, with a view to see whether we cannot penetrate the mystery a little further, or at all events see our way to undertake further inquiries.

There are, I think, good reasons why we should at times restrict ourselves, in the main, to facts which we observe at home. In instituting a comparison between different centres, there is, to start with, the difficulty that under summer diarrhœa we may not be including the same disease or diseases in different localities. The conditions are so widely different socially that any conclusions as to the effects of other phenomena must be greatly weakened, notwithstanding that each community constitutes a complete population. Then, again, some towns are placed at a considerable elevation, on the Pennine range. Not only are the temperatures of air and soil thereby lowered, but there is a greater rainfall than in lower localities. The impression is liable to be thus produced or, at least, intensified that a high rainfall goes along with a low diarrhœal rate. Even where the system of removal of excreta is a conservancy one, and similar in character in two places, the structural and other arrangements may be so much better in one place as greatly to diminish the evils incidental to such a system in another. Then, again, the soil of elevated situations is likely to be hard rock, and quite impermeable, so that an undue impression is created that diarrhœa is

associated with a porous subsoil. From the data, however, obtained for Manchester no such difference of incidence in fatality of diarrhœa, as between one kind of soil and another, can be deduced. It is not denied that such a relation may exist, but the evidence does not appear sufficient to establish the thesis.

For such reasons as these it is a great advantage, in forming conclusions as to the explanation of facts, to deal with areas all the characters of which are quite familiar to the reasoner. The atmospheric temperature, the temperature of the soil, and the rainfall are approximately the same throughout.

There have been not a few inquiries into this subject, some of which, like Dr. Ballard's and Dr. Newsholme's, have dealt with all the aspects of the subject. Others, again, like Professor Delépine, have concerned themselves rather with special aspects of it. Professor Delépine adduces evidence showing that milk may have much to do with the spread of the disease, as it arrives from a distance in towns. Dr. Newsholme, on the other hand, from the observed facts in connexion with breast-fed children, with infants fed on condensed milk, and from comparison of the fatality experienced by infants fed on milk brought from a distance with that experienced by those fed on milk produced near the town, came to the conclusion that the infection experienced was domestic. Notwithstanding the striking facts adduced on the other side, I now feel obliged to concur in the latter view, in support of which it may be pointed out that diarrhœal mortality does not rise in June, notwithstanding the exposure of outside milks to high temperatures—before a certain period is reached.

Summer or epidemic diarrhœa is a term applied to an affection marked by a somewhat definite group of symptoms, in which vomiting sickness, copious diarrhœa, rice-watery and green stools, and finally convulsions play a conspicuous part. This condition is not rarely somewhat prolonged, and is often attended with some degree of fever. On the one hand it shades into typhoid and paratyphoid fevers, and on the other it is not rarely the termination of a tuberculous enteritis or some wasting affection. From the study of a large number of histories, Ballard concluded that he was dealing with a definite disease, and that is also my opinion from the histories of illness which have been obtained for me. Yet I have no doubt that enteric fever in infants may be, and not rarely is, mistaken for diarrhœa. The grounds for this opinion will be readily seen on referring to the histories of enteric infection given in the annual reports on the health of Manchester.

Whether summer diarrhoea is produced by one definite micro-organism or is an illness conditioned by several allied bacilli, it is a clinical entity pursuing a very definite course, and as such is susceptible of study. One of the advantages of an intensive study is the uniformity secured in the deaths classed as "summer diarrhoea." In Manchester, since Dr. Tatham held office, in any case where doubt could exist the certifying practitioner has been asked by letter whether in his opinion the death should be regarded as due to epidemic diarrhoea, and it has been entered accordingly. There have been several claimants for the post of director of the fatal summer outbreak, of which the chief are Gaertner's bacillus, a similar organism isolated by Professor Delépine, and the *Bacillus enteritidis sporogenes*. I am not in a position to pass a judgment on this question. It appears not improbable that the most malignant forms of the disease are those which survive the winter and spring, and serve as foci from which smaller doses of infection are distributed, a process which leads to attenuation of the severity, and also to prolongation of the disease. Ballard adduced abundant proof that the few fatal cases which occur week by week before the summer ascent represent a large number of attacks at different ages, so that at the commencement of the annual ascent there are abundant foci from which the disease can be propagated. Between infancy and old age, however, the fatality is slight, so that its widespread distribution escapes attention. As a rule, also, the sufferer is able to go out to the closet, a fact which may prove to have considerable significance.

Epidemic diarrhoea is infectious. If the history of institutions were faithfully recorded, it would probably be found that it not infrequently spreads in these. The course of its summer fatality is that of a rapidly spreading disease, transmitted by some specific agency. When, as in Manchester in 1904 and 1905, a number of individual cases are investigated, infection from person to person becomes highly probable in a considerable proportion of the cases, the agency being often left vague. It is true this is associated with a low fatality in the winter, spring, and early summer. Nevertheless diarrhoea is kept well alive through great part of the year, in spite of the circumstance that there appears to be no agent capable of conveying it from one house to another. From the annual report for 1908 I insert a table (p. 135) showing the distribution of mortality in the various sanitary districts of the city for a period of thirteen years.

It will be seen that these districts frequently change the order of their infantile rates of mortality, that some of them are subject to great

fluctuations, and that the privy-midden districts, like Bradford and West Gorton, and Clayton, are apt to predominate in seasons of high diarrhœal mortality. All these features mark out the disease as being of an infectious character. The further discussion of the facts will tend to lay stress on the influence of the house fly, and it will be found that the course of enteric fever has an important bearing on the subject. But it will be convenient to give at this point a brief account of the efforts which have been made in successive years to elucidate the course of the disease.

From the inquiries of Boobyer, Scurfield, Tattersall and others, we may take it as proved that there is an intimate relation between the storage of excreta in privy-middens and a high diarrhœal mortality. The materials at my disposal have not permitted me to adduce precise evidence on this point. It suffices, however, to note that diarrhœal mortality is excessively high in all those sanitary districts of Manchester in which the privy-midden has prevailed, as in Bradford, Clayton, Openshaw, and West Gorton. This is also the case in the newly-added district of Gorton. Moreover, the mortality in these districts increases relatively in years of high diarrhœal fatality. On the other hand, in the more central portions of Manchester in which pails have long replaced middens, owing to defects in the structural and other arrangements, there has been much nuisance from the recesses in which the pails stand. Thus, although the contents of the pails are taken away once a week, and no generation of flies can occur in their interior, I am informed that the pupæ of the smaller house fly have been found in these recesses. It is probable that much improvement in this respect has taken place in recent years. But there exist at present quite sufficient materials in collections of horse manure, and of other organic matters, to provide for the continued production of flies, even when all the pail-closets have become innocuous.

The social condition of the population has also much influence on the diarrhœal death-rate. Where the people are very poor, the children are also disproportionately unhealthy at the outset, and the disproportion rapidly increases after birth, owing chiefly to ignorance or carelessness on the part of many mothers.

If reference be made to the table (p. 135) it will be seen that the death-rate is persistently highest in the central and poorest sanitary division, and then in South Manchester, the next poorest. It is true, the differences between the death-rates in different parts of Manchester are not so great as in the case of phthisis, and it is manifest that other

TABLE I.—DEATHS AND DEATH-RATES FROM DIARRHEAL DISEASES IN THE VARIOUS DIVISIONS OF THE CITY, WITH DEATH-RATES UNDER ONE YEAR PER 1,000 BIRTHS, FOR 1896 TO 1908, AND AVERAGE FOR TEN YEARS, 1898 TO 1907.

	1908		Death-rates under one year per 1,000 births														Average 10 years	1908
	Estimated population	Deaths	Death- rates	1896	1897	1898	1899	1900	1901	1902	1903	1904	1905	1906	1907			
City of Manchester	648,846	591	0.90	24.8	39.1	46.3	63.7	35.6	47.5	13.0	22.1	34.1	30.8	39.8	12.2	34.5	24.0	
(1) Manchester township	125,197	201	1.58	30.0	45.4	54.6	78.5	47.5	61.6	16.6	31.3	40.9	47.7	58.5	18.0	45.5	37.8	
(2) Northern districts	197,527	134	0.67	19.6	34.8	37.3	57.4	24.4	42.3	10.7	15.0	30.1	31.3	30.8	10.3	29.0	17.8	
(3) Southern districts	326,122	256	0.77	24.3	37.7	46.7	58.7	36.1	43.2	12.4	22.0	33.0	22.5	36.7	10.7	32.2	21.5	
Manchester township:																		
Ancoats	43,206	77	1.76	30.7	58.3	45.3	85.0	48.6	57.4	17.1	30.2	35.4	50.9	54.9	21.0	44.6	40.8	
Central	24,922	41	1.62	46.6	45.2	75.2	71.1	55.0	66.1	15.0	48.5	51.0	52.4	54.9	19.1	50.8	41.3	
St. George's	57,069	83	1.43	20.8	36.0	52.8	76.7	43.4	63.3	16.9	25.5	41.1	43.3	62.8	15.3	44.1	34.3	
Northern districts:																		
Cheetham	42,376	16	0.37	20.1	22.5	22.6	36.0	18.0	27.3	9.4	10.1	10.7	18.8	19.6	7.5	18.0	10.9	
Crumpsall	9,430	3	0.31	16.1	26.3	20.4	60.9	14.6	23.2	14.9	9.8	15.2	23.9	20.7	—	20.4	9.3	
Blackley	9,830	3	0.30	4.7	4.4	9.8	44.2	4.4	9.2	4.0	12.1	23.5	3.8	3.5	3.7	11.8	6.9	
Harpurhey	23,318	7	0.30	24.7	42.6	44.7	72.6	11.3	36.5	1.8	15.9	13.1	21.7	40.1	13.7	27.1	11.7	
Moston	20,826	1	0.05	9.1	17.5	51.0	29.1	2.8	19.0	11.7	8.8	25.5	8.4	13.2	2.0	17.2	1.9	
Newton Heath	39,153	38	0.96	15.7	32.9	37.8	57.9	25.4	49.7	12.3	15.6	31.4	43.3	36.0	16.8	32.6	22.0	
Bradford	25,355	40	1.55	24.9	49.8	58.1	93.3	43.3	62.7	13.2	26.9	52.5	47.7	53.6	8.7	46.0	34.6	
Beswick	12,588	20	1.56	20.3	52.5	23.6	46.4	40.6	50.0	20.6	8.4	60.8	43.2	23.0	19.0	33.6	37.6	
Clayton	14,651	6	0.40	41.4	55.2	61.6	66.7	36.4	94.9	6.2	20.2	38.4	40.1	41.2	7.8	41.4	9.8	
Southern districts:																		
Ardwick	45,324	42	0.91	19.2	27.7	50.2	61.7	43.7	48.4	11.5	20.0	38.5	28.7	42.8	12.8	35.8	29.0	
Openshaw	29,040	35	1.19	25.7	42.6	58.5	64.3	44.7	48.1	14.2	27.4	28.0	23.9	50.0	8.2	36.7	24.7	
West Gorton	32,316	24	0.73	21.4	42.7	69.3	85.7	52.6	58.4	21.2	31.1	47.2	35.1	60.0	28.5	49.0	19.6	
Rusholme and Kirk- manshulme	27,007	13	0.47	17.2	26.3	43.7	34.0	15.4	33.3	11.6	14.0	12.7	10.5	21.2	2.4	19.9	11.1	
Chorlton-on-Medlock	55,597	42	0.74	28.7	24.2	32.8	58.7	25.1	17.9	7.1	23.9	23.8	29.1	32.8	13.7	27.5	25.9	
Hulme	62,629	87	1.37	26.3	50.3	40.6	49.5	33.1	43.6	12.0	18.7	39.2	22.3	33.6	10.8	30.3	28.3	
Moss Side	28,522	2	0.07	—	—	—	—	—	—	—	—	—	—	—	3.5	29.8	1.6	
Withington	45,687	11	0.24	—	—	—	—	—	—	—	—	—	11.2	19.4	2.2	10.9	10.3	

¹ Average for three years.

factors besides the social have an important influence in producing it. Nevertheless, this factor is conspicuous no less in the central than in other districts, such as Bradford, Ardwick, and Openshaw. How great this influence is can only be determined by direct inquiry into the health of fatal cases of diarrhœa prior to the attack. The result of such inquiries is given in the annual reports for 1905 and 1906, and it is shown not only that the great majority of the infants were in poor health before diarrhœa set in, but that the social circumstances of a considerable section were of a miserable kind. This is not an isolated experience. Dr. Hope has similarly found a close association between diarrhœa and dirty home conditions, and has pointed out that fatal diarrhœa tends to recur in the same household. It would, however, be possible to overestimate the significance of this fact, since the absence of breast feeding has much to do with diarrhœal fatality, and this also tends to recur in the same household. Again, a large minority of healthy infants contract fatal summer diarrhœa, and it is this fact, and the nature of the individual occurrences, which convey a direct and strong impression that summer diarrhœa is infectious. Further, in spite of the absence of middens, taking into account the large number of pail-closets, of stables, and of collections of refuse of one kind or another, it is doubtful whether one can say that the central districts are essentially freer from those causes which one believes to be concerned in spreading diarrhœa than were the outlying privy-midden districts. The effect of wretched home conditions is demonstrated more by the persistence of a steady high mortality than by its average magnitude.

Dr. Hope has estimated that artificially-fed infants suffer more from fatal diarrhœa in the first three months of life than do breast-fed infants in the proportion of fifteen to one. The proportion is at least as high as this, according to our inquiries.

The explanation of this disproportion is not quite simple. It is usually taken to mean that infants artificially fed are much more exposed to infection than breast-fed infants, and this, no doubt, is true. But we must, also, believe that breast feeding renders children less susceptible to attack when exposed to infection, and less likely to die when infected. Direct infection may be conveyed in a variety of ways apart from the food, as by rubbing the gums of the infant, the use of comforters, placing the infants on dirty floors and sofas, and in other ways. We are thus able to see how it is that a certain proportion of breast-fed infants may contract diarrhœa directly, even if flies be not reckoned with.

The essential problem in summer diarrhœa, however, is the summer

wave, ascending as it does steeply, and descending with but little less of abruptness. It is to this period that the fatality is due. Broadly speaking, it corresponds to a similar upward movement and descent of the temperatures registered at a depth of 4 ft. in the soil. The readings of the air thermometer in the shade and of the 1-ft. earth thermometer do not correspond closely to the course of this wave of deaths, being subject to considerable fluctuations. But it is now generally agreed that the mortality must have closer direct associations with the surface than with the deeper temperatures. Many attempts have been made to penetrate into the nature of the supposed association between the conditions of the soil and the rise of summer diarrhoea, but not with much success. It has been conjectured that under favourable conditions of temperature and moisture growth takes place at a particular period of the year of those micro-organisms which cause diarrhoea. These have been variously supposed to start near the surface, and at a considerable depth. Reaching the surface, they are supposed to be subject to drought, but not killed. Subsequently wafted into the air, these germs, it is supposed, may reach food, which they contaminate.

If the figures be written down showing the number of cases of enteric fever commencing week by week, a curious phenomenon is noticed, which at an early period arrested my attention—viz., that in some one week of the year the number of cases makes a sudden leap upwards, an ascent afterwards maintained, it may be with fluctuations. This fact has not been in evidence in every year, though in most it has. An effort was made as far back as 1898 to furnish an explanation of this rise from the meteorological data, and it was observed that it was associated with a preceding rainfall, lying between two dry, warm and sunny periods, although the critical rainfall, as I called it, did not occur at a fixed interval before the critical rise of cases. Moreover, the rainfall in question was always during ascending temperatures. The conditions in question were such as would be likely to produce a great increase in flies, but no such idea suggested itself to me in the earlier years. Nevertheless this peculiar rise in the enteric curve was pursued through a series of years, although the explanation eluded discovery.¹

It was felt that this phenomenon was the key to the explanation of the autumnal rise of enteric fever, and such is still my impression. Moreover, it might very well be that the autumnal rise of diarrhoea would be elucidated when the behaviour of enteric fever was explained.

¹ Manchester Annual Reports, 1898, *et seq.*

Flies first came prominently into notice in connexion with enteric fever in the American-Spanish campaign, when they were spoken of confidently as transmitting infection from the excreta of the sick to the healthy. English medical observers in the Boer war were strongly of the same view. Dr. Arthur Newsholme, Dr. Nash, and other observers, amongst whom was my neighbour Dr. Martin, medical officer of health for Gorton, arrested by these observations, directed attention to the agency of flies as possible transmitters of the infection of diarrhœa and other diseases. It was not, however, till 1903 that I began to make observations on the subject, nor until 1904 that systematic captures of flies were made.

Before proceeding to discuss the results of these observations, it is necessary to consider the hypotheses on which such a phenomenon as the course of the diarrhœa annual wave can be explained.

We may first consider the influence of the soil. In favour of the view that under some unknown conditions the soil may have to do with the propagation of diarrhœal diseases, we have such facts as these :—

(1) Dr. John Robertson's investigations, in the course of which he showed that, under special conditions, cultures of the typhoid bacillus planted in soil could survive the winter and be recovered from the ground.

(2) Professor Delépine's demonstration of the persistence of viability of typhoid bacilli in the walls of a privy-midden for a whole year.

(3) Occasional attacks of enteric fever in men engaged in drainage work (Manchester histories).

On the other hand, from the work of Dr. Sidney Martin it is to be inferred that under all ordinary conditions cultures of typhoid bacilli planted in the soil are speedily overgrown, and the bacillus is soon irrecoverable.

Supposing, however, that enteric bacilli and similar micro-organisms can grow in the soil, the application of this circumstance to the course of diarrhœa is by no means easy. This disease pursues its even course independent of drought or rain, unless the latter is so great as to greatly lower the temperature. It is difficult to conceive of the growths in question spreading upwards from a considerable depth; it is more difficult to conceive of them escaping from the surface, wet or fine; it appears certain that, if such a growth did occur, it would be confined to a comparatively short period, corresponding to the periods occupied in the production of laboratory cultures. There is thus an inherent improbability that such growths would occur only at one particular

season, especially in the case of the typhoid bacillus (which is not very exacting as to the temperature which it must have), failing with similar conditions of surface temperature outside this special season. It is excessively unlikely that the interruptions to the escape from the soil of bacilli necessarily produced by wet weather would not affect the course of diarrhoea. It is difficult to see how bacilli are to escape from the soil in the well-paved central portions of towns, or to see why they should fail to be propagated and be distributed from the comparatively uncovered parts of outlying districts. If the course of diarrhoea depended on micro-organisms growing in or on the soil, it would participate in the fluctuations of rainfall and temperature, which would certainly affect such growths. This it does not. One is driven, I think, to abandon the idea that the growth of bacteria, whether in or on the soil, has to do with the annual wave of diarrhoea.

Other hypotheses have been put forward. One is that fruit may be responsible. But, in the case of diarrhoea, the disease appears first in the infant in house after house, and it is certain that the infant has no fruit; the effect produced by fruit on the annual course of the disease can therefore only be very partial. Nevertheless, if flies can infect infants' food they can infect other food, and fruit in particular, so that the histories of direct exposure of infants attacked to previous diarrhoeal infection becomes important.¹

Another view is that heat may be itself the cause of the disease, or, if not of the disease, at any rate of the fatality. As against this explanation we must adduce the comparative immunity of the infants of better-off households, the escape of artificially-fed children when sufficient care has been expended on their nurture,² and the comparatively slight incidence of fatal diarrhoea in many towns in the south, in spite of high temperatures. Yet it is impossible absolutely to refute the influence of temperature. The above arguments suffice to show that diarrhoea does not arise from the action of heat in furthering some infection pre-existing in the child, but they do not remove the possibility that heat acts injuriously on weakened organisms, predisposing them to the reception of infection. Yet, clearly, heat can in no way account for the rapid spread of the disease among particular classes of the population.

In his Milroy lectures, again, Dr. Waldo suggested that the spread of the disease was due to dust, and was strongly inclined to favour the idea

¹ See Manchester Annual Reports for 1904 and 1905 and Dr. Sandiland's inquiry.

² Dr. Matthew Hay, Health of Aberdeen, 1908.

that the specific *materies morbi* came from horse-dung. There does not appear, however, to be much more in favour of one view than of the other. There is practically no doubt that the latent period of fatal diarrhœa does not often exceed a week, and the most common period is probably two or three days. The course of fatal diarrhœa should be seriously affected by heavy rain lasting several days if dried horse-dung were the cause ; yet diarrhœa and flies pursue their way with comparatively little disturbance, wet or fine. Dr. A. H. Ainsworth, in a series of curves, shows the enteric waves rising out of the heavy rains of the monsoon at Poona, and sinking back before the rains are over. The excess of moisture is, in that region, doubtless necessary for the development of the house fly, and it breeds accordingly, having its annual ascent in the same period. Unfortunately, there is only one curve of flies given, but one gathers from Dr. Ainsworth's paper that this is the usual course of events. To come nearer home, why should dust effect so much in August, so little in March, April, May, June, and July ? There is, in effect, no disturbance in the diarrhœal fatality till flies begin to multiply. We must, I think, set aside transmission by dust as palpably inadequate as an explanation.

What we require for the explanation of the facts of summer diarrhœa is the presence of some transmitting agent rising and falling with the rise and fall of diarrhœa, the features pertaining to which must correspond to and explain the features of the annual wave of diarrhœa.

None of the other factors of which we have cognizance do afford such an explanation, and we come by exclusion to consider the house fly. The process of conveyance of infection is not striking and arresting, as it is in military camps abroad ; nor does the number of flies usually approach that observed in tropical and subtropical countries. We are therefore obliged to attack the question *de novo*, and examine such evidence as we possess to see whether we may rest reasonably confident that in flies we have found the transmitting agent sought for.

If the house fly is the transmitting agent in summer diarrhœa, the following conditions should be fulfilled :—

(1) (*a*) There should be evidence that the house fly carries bacteria under the ordinary summer conditions ; (*b*) house flies should be present in sufficient numbers in houses invaded by fatal diarrhœa.

(2) There should be a close correspondence between the aggregate number of house flies in houses and the aggregate number of deaths from diarrhœa week by week.

(3) The life-history of the house fly should explain any discrepancy between the observed number of flies and the observed number of deaths.

(4) The minority of breast-fed children not apparently accessible to infection should receive explanation.

(5) There should be a closer correspondence of diarrrhœal fatality with the number of flies than with any other varying seasonal fact.

(6) Any other closely corresponding seasonal fact should be capable of interpretation in terms of the number of house flies.

(7) Any variation from district to district in the annual curve of deaths should be accompanied by a similar variation in the curve of flies.

(8) It will be at once manifest when we come to enteric fever that the house fly plays but a minor direct part in the production of the annual wave. Such part, however, should have reference to the number of flies and of pre-existing centres of infection. If it can be shown that that portion of the enteric wave which is connected with flies changes from one period of time to another in such a manner as to be explainable in terms of flies but not of meteorological conditions, the evidence in favour of flies will be greatly strengthened.

(9) No other available hypothesis must be capable of explaining the course of summer diarrrhœa.

In working out the above statement it will not be possible to adhere to the order of these propositions, which, however, will be kept steadily in view.

I come now to discuss the observed relation of the house fly to diarrrhœa in Manchester. In any consideration of this question the first need is to ascertain that a relation does exist. Accordingly, after a preliminary trial in 1903, we proceeded in 1904 to select a number of houses in which it was thought that the householder could be relied upon to carry out continuous and careful counts day by day of the number of flies caught. These observers were provided with bell-glass traps covered above but accessible from beneath, the lower rim curving inwards to form a semicircular ring, into which was to be poured day by day a thin sweet beer, with which the observers were provided. This beer is very attractive to flies; they enter from below, hasten to partake of the beverage, and get drowned. It might be supposed that they are attracted simply by the moisture. This, however, is not so. I am informed that other alcoholic beverages exercise a similar attraction for them, and I am told by one householder that in the capture of cockroaches his success with the "demon" trap has been much increased by pouring a little whisky into the cup of the trap.

I prefer this trap to stick papers. It is true no distinction between *homalomyia* and *musca* can be made out in flies thus captured. The

same applies in great measure to stick papers. With balloon wire traps our captures have been scanty and unrepresentative. The beer trap gives a fair measure of numbers, if properly used, and the flies are easily counted. It is possible that the kinds captured by the different traps vary in their numerical proportions, and I regret that, for reasons of expense, we substituted in 1909 stick papers for beer traps. For the purpose of distinction, balloon traps are satisfactory, the flies being preferably killed by chloroform.

The flies captured, musca and homalomyia together, are emptied out at the end of twenty-four hours, and counted. The number is then entered in a book by the observer, the trap cleaned out, and recharged with beer. At the end of the season the individual enumerations are entered in a record book, day by day, and are then collected in weeks for each station and for all stations. In this procedure, during the five years of completed observations, 1904, 1905, 1906, 1908, and 1909, there has been no substantial alteration except the alteration in traps in 1909. In the various years now one, now another station has been added, other than private houses, including the depots of the Cleansing Department, Monsall and Clayton Hospitals, and Mill Street Police Station. The observations made at these, however interesting in themselves, appear to introduce an element of confusion from the excessive numbers liable to be captured at particular times, which do not agree with those of smaller house stations, and which cause the total curve to be unrepresentative and irregular. When the numbers captured at the different stations are added up week by week, the weekly numbers form for each station a series which is subject to considerable fluctuations. These numbers show often two maxima, sometimes three. Even when the numbers are very large the maxima for different stations do not coincide. It is only when the numbers are added up week by week in all the stations that we get a coherent and graduated curve. It is evident that to get a reliable and complete representation of the numbers of flies visiting or present in houses throughout the season we should require a vast number of stations, and in each a reliable observer. Our completed curve, especially with a small number of stations, must therefore be imperfect as a representation of the total distribution of flies. In this respect the observations carried out in London are much more satisfactory, at least in numbers, than ours. The stations are in the London observations grouped round certain centres specially attractive to flies. Some of these, however, attract flies for purposes of feeding, others for the purpose of breeding. The distinction is fundamental. The

former will attract the maximum of flies early on before the maturity of the sexual organs, the latter partly no doubt for feeding purposes, but largely also for reproductive reasons. Now, a fly is not sexually mature until three weeks or so after its emergence from the pupal stage. The maxima may therefore well diverge by three weeks or more.

For ordinary average purposes of estimation house observations with but little reference to special centres of attraction will probably give quite useful results. As a matter of fact, of course, owing to the wide distribution of stables, such centres represent the stable interest chiefly; that is to say, they represent flies in their youth, moving from house to house, maturing, feeding, the element with which we are chiefly concerned, as well as the mature flies which have deposited their eggs. A sweet factory does not necessarily augment the numbers in houses near it. It may, and probably often does, deplete them of their migratory elements. The flies come, of course, primarily from collections of horse manure, of domestic refuse, and so forth.

It is probable that flies are *musca* or *homalomyia* according as the materials in which they are produced ferment or not, with consequent elevation of temperature. *Musca* probably is produced chiefly in horse manure, fermenting tips, and in middens when the ground temperature is high. *Homalomyia* is produced, also, in the recesses of pail-closets, in vegetable refuse, in the shallower parts of tips, and in middens before fermentation sets in—i.e., before the ground temperature rises. There is no reason to suppose that *homalomyia* exercises any special influence on the spread of disease. Diarrhœal fatality does not ascend in May or June, when *homalomyia* has already attained considerable numbers. On the other hand, there is no reason for believing that this fly does not take its fair share in the spread of the disease. In any case its relatively small numbers, when diarrhœa is most prevalent, necessarily relegate it to a secondary position.

No substantial error is involved in adding *muscæ* and *homalomyia*.

As we have seen, the results obtained by adding the numbers enumerated at a limited number of stations are necessarily imperfect. Fortunately we are able to apply some corrections, partly from the facts themselves, partly by the use of first approximations. To this subject I shall afterwards return.

It scarcely seems necessary at this point to elaborate much the consideration of the course of the numbers of flies captured week by week; it must suffice here to point out that the production of fly swarms on which the numbers in houses must depend is subject to many influences.

If the collection of manure or refuse is covered over, it depends on the retentiveness of moisture of the manure and the speed with which the top and sides are buried. Multitudes of larvæ perish for lack of moisture; many are destroyed by beetles. Often when the pupæ reach maturity the flies produced must be unable to struggle through the overlying material. Small, open collections of horse manure must be easily chilled by rain, and fermentation arrested; the production of flies in these is more difficult than in large collections, and no doubt requires warmth of soil and of atmosphere in excess of what suffices for large collections. The number of horses in proportion to the size of the manure heap must be an important factor; if the number of horses is disproportionately large, the flies will not be able to emerge except at the sides, and so forth. The fertility of flies is by no means much in excess of the requirements; the production of swarms is largely, in fact, the result of happy accident. It may happen, for example, that the distribution of a large manure heap is eminently favourable to the production of a swarm of flies, and that the weather is particularly favourable; meantime the farmer's cart arrives and transfers elsewhere the struggling brood. A spell of wet weather arrives, chills and kills the flies about to emerge in shallow collections of refuse, but supplies much-needed moisture for development and fermentation to large heaps, or keeps alive the larvæ in covered collections. The influence of weather conditions is complicated and not easily calculable.

It will be necessary for a fuller understanding of the facts to consider some details of the life-history of flies, but we may now proceed with the summary of the facts. I give, therefore, for five years the number of deaths week by week, the number of flies captured in our stations in the same periods, the mean atmospheric temperatures, and the mean temperatures at depths of 1 ft. and 4 ft. For three years are given also the numbers of fatal attacks of diarrhœa commencing in those weeks. There are, however, two serious drawbacks to these series of cases arranged in weeks of commencement. Many families in which a fatal attack has occurred had disappeared at the time of inquiry, and in many others the period of commencement could not be accurately ascertained; the series is therefore in each case imperfect. It is scarcely necessary also to renew the caution that the diagnosis is subject to considerable deduction; it is, however, necessary to remember that this deduction is much more serious at the commencement of the curve than at its height, owing to the smallness of the numbers. It is with these imperfect data, however, that we have to work.

OBSERVATIONS ON HOUSE FLIES.

The data for the five years may be presented as shown on p.p. 146-149.

These data are illustrated by charts, which require some explanation. The charts are divided up as usual. In the case of flies, that number is taken as unity at which fatal cases begin to increase. The numbers for 1904, 1905, and 1906 are given on p. 160; and the numbers in other weeks are reckoned as multiples of these. In years in which this is not easily determined, 2,000 flies are taken as unity for fourteen or sixteen bell-traps; ten cases or ten deaths are taken as one for the corresponding curves. Temperatures are reckoned by taking the lowest mean weekly atmospheric temperature, subtracting from the mean weekly temperature under consideration and dividing the difference by two. We note the absence of direct relation between the curves of temperature or rainfall and those of flies or deaths from diarrhoea; also the manner in which the curve of fatal cases or deaths falls away from the curve of flies in the middle of the decline. This is probably due, chiefly, to immobilization of flies from cold and fungus. We note, also, that the number of cases commencing in weeks is much smaller than the number of deaths, only those deaths being included in which the parents could be found, and the date of illness ascertained with tolerable precision. The commencement, summit, and end of the annual wave furnish information as to the average interval between attack and death in infants.

In 1904 and 1906 the maximum of attacks precedes by a fortnight, in 1905 by a week, the maximum of deaths. If, however, we average the attacks and deaths in successive weeks, the interval of the resulting curves becomes a week. We may conclude that the average period is greater than a week and less than a fortnight, being nearer to the former period than to the latter. We may, also, for any year, start from the dates of attack, and record the number of deaths occurring within a week of attack and in successive weeks thereafter. Doing this for the year 1908, we find the following intervals between attack and death:—

		Week of attack	Second week	Third week	Fourth week	Fifth week	Sixth week	Seventh week	Eighth week	Ninth week	Tenth week +
Deaths in											
First period	...	19	9	5	1	7	—	—	—	—	7
Second „	...	73	37	11	9	3	2	1	1	1	2
Third „	...	82	73	55	17	18	8	1	3	5	14
Total		174	119	71	27	28	10	2	4	6	23

We thus see that, as the diarrhoea season advances, the length of the fatal illness is increased. In the maximal week of illness of the cases supposed evenly distributed over the week, three-sevenths would die in the following week.

146 Niven: *Summer Diarrhœa and Enteric Fever*

1904

Week ending ..	July 9	July 16	July 23	July 30	Aug. 6	Aug. 13	Aug. 20
Enteric cases	3	3	6	6	9	8	7
Deaths from diarrhœa	5	—	14	48	77	91	107
Fatal cases commencing	14	23	40	70	120	103	53
Mean temperature in shade	59·8	66·6	66·2	64·0	66·5	59·1	57·1
Rainfall in inches	0·235	0·230	0·090	0·420	0·630	1·090	1·015
Underground temperature at 1 ft.	59·7	63·8	64·1	64·5	65·0	62·6	60·1
" " 4 ft.	55·7	56·9	58·6	59·7	60·2	60·9	60·5
Number of flies caught in 12 bell-traps	1498	4039	5234	6699	19081	18440	8537

1905

Week ending ..	July 6	July 15	July 22	July 29	Aug. 5	Aug. 12	Aug. 19
Enteric cases	1	1	—	7	2	4	9
Deaths from diarrhœa	7	7	22	62	81	89	67
Fatal cases commencing	12	39	58	70	72	61	51
Mean temperature in shade	61·3	67·8	63·2	61·8	59·8	58·6	60·1
Rainfall in inches	—	0·830	0·780	0·780	0·970	0·625	1·050
Underground temperature at 1 ft.	63·1	65·7	65·4	64·6	61·3	60·6	60·9
" " 4 ft.	57·8	58·9	60·2	60·9	61·0	60·1	59·9
Number of flies caught in 12 bell-traps	2788	4456	7799	9493	9627	8542	7112

1906 In this year the curve of cases may be neglected owing to faulty investigation at the height of the epidemic. This is clear from the numbers.

Week ending ..	July 7	July 14	July 21	July 28	Aug. 4	Aug. 11	Aug. 18
Enteric cases	9	4	5	3	7	2	6
Deaths from diarrhœa	4	6	6	12	17	34	80
Fatal cases commencing	9	3	20	44	74	81	68
Mean temperature in shade	60·4	57·1	59·1	63·4	64·9	63·0	59·6
Rainfall in inches	0·070	0·440	0·750	0·340	0·270	0·640	1·220
Underground temperature at 1 ft.	60·2	60·1	59·8	62·4	63·9	63·6	61·8
" " 4 ft.	56·4	57·1	57·2	57·8	58·7	59·7	59·9
Number of flies caught in 17 bell-traps	3489	4909	7852	9280	12144	16101	16303

1907

Week ending ..	Aug. 10	Aug. 17	Aug. 24	Aug. 31	Sept. 7	Sept. 14
Enteric cases	2	4	3	2	1	4
Deaths from diarrhœa	8	9	5	3	5	12
Fatal cases commencing	4	10	—	9	9	16
Mean temperature in shade	59·4	59·1	55·8	57·1	54·8	60·9
Rainfall in inches	1·020	1·660	0·760	0·080	0·790	—
Underground temperature at 1 ft.	59·4	59·1	57·7	56·8	55·7	57·3
" " 4 ft.	57·0	57·2	57·3	57·0	56·6	56·1

Aug. 27	Sept. 3	Sept. 10	Sept. 17	Sept. 24	Oct. 1	Oct. 8	Oct. 15	Oct. 22	Oct. 29	Nov. 5	Nov. 12	Nov. 19
9	6	10	13	7	18	7	7	4	7	9	5	7
85	68	43	36	28	24	23	11	3	3	4	1	—
34	32	18	23	13	10	7	1	—	—	—	—	—
54.7	62.3	57.7	57.7	57.0	53.4	49.8	49.5	55.5	49.1	50.0	46.9	45.8
1.530	0.590	0.350	0.145	—	0.595	0.475	0.125	0.790	0.060	0.095	1.825	0.170
57.0	61.3	59.0	56.9	55.3	53.8	51.3	49.8	51.2	49.8	49.2	48.1	45.6
59.5	58.6	58.6	59.3	57.7	56.9	55.8	54.6	53.5	53.1	52.4	51.9	51.0
6609	9802	6837	5485	5500	3642	2398	2070	2214	—	—	—	—

Aug. 26	Sept. 2	Sept. 9	Sept. 16	Sept. 23	Sept. 30	Oct. 7	Oct. 14	Oct. 21	Oct. 28	Nov. 4	Nov. 11	Nov. 18
8	8	6	7	11	7	13	6	12	9	10	4	10
81	61	61	42	20	15	10	9	5	4	6	3	3
37	21	12	12	8	4	—	—	—	—	—	—	—
59.1	56.1	58.5	53.4	54.1	56.4	48.1	49.7	40.8	42.2	46.4	45.0	39.2
1.580	0.850	1.510	0.330	—	0.140	0.970	0.830	0.510	0.390	0.640	0.922	0.040
59.9	59.0	58.5	55.3	54.1	52.5	50.2	51.8	45.0	41.2	43.9	43.5	42.1
59.6	59.1	58.7	58.0	56.9	55.8	54.8	53.6	52.6	50.4	48.4	48.0	47.4
6112	5309	5138	3815	2978	1380	—	—	—	—	—	—	—

Aug. 25	Sept. 1	Sept. 8	Sept. 15	Sept. 22	Sept. 29	Oct. 6	Oct. 13	Oct. 20	Oct. 27	Nov. 3	Nov. 10	Nov. 17
4	4	13	20	19	14	13	22	16	23	16	7	14
102	104	135	136	86	58	35	28	11	13	16	9	5
88	65	28	20	17	12	4	5	—	—	—	—	—
62.5	66.8	63.9	56.6	56.3	52.9	57.5	56.3	46.6	52.8	45.5	46.2	43.5
0.920	0.010	0.520	0.990	0.050	—	0.790	0.810	2.270	0.780	1.020	0.150	1.030
61.5	62.3	61.6	59.2	56.9	53.6	53.9	55.8	50.7	50.2	45.5	45.5	42.9
59.9	59.9	60.1	60.2	59.3	58.1	56.6	56.0	55.7	54.4	53.1	51.5	50.1
16605	23572	23144	19747	13421	10651	11976	6873	2685	3349	—	—	—

Sept. 21	Sept. 28	Oct. 5	Oct. 12	Oct. 19	Oct. 26	Nov. 2	Nov. 9	Nov. 16	Nov. 23	Nov. 30
2	7	3	10	6	6	6	10	7	13	12
18	21	32	45	28	18	12	4	5	5	1
26	25	41	10	12	5	1	4	2	2	2
58.1	60.7	55.7	52.9	50.6	49.6	49.3	50.1	45.4	42.5	41.2
—	—	0.180	1.175	0.670	0.980	0.345	0.150	0.495	0.935	0.760
57.3	56.2	55.8	53.1	51.2	51.2	49.8	48.0	48.7	46.3	43.5
56.2	56.3	56.1	55.9	55.9	55.0	53.9	52.6	51.7	50.9	49.9

1908

Week ending ..	July 11	July 18	July 25	Aug. 1	Aug. 8	Aug. 15	Aug. 22
Enteric cases ...	—	1	1	1	2	2	10
Deaths from diarrhœa ...	6	9	20	22	48	82	70
Fatal cases commencing ...	11	21	28	54	65	48	45
Mean temperature in shade ...	59.1	58.2	62.8	60.4	61.6	57.6	58.2
Rainfall in inches ...	1.985	2.545	0.255	0.010	—	0.240	0.700
Underground temperature at 1 ft. ...	61.0	59.3	61.0	62.3	62.1	60.5	59.2
" " 4 ft. ...	57.3	57.7	57.8	58.4	59.0	59.3	59.1
Number of flies caught in 15 bell-traps, in houses only ...	6959	6635	9579	19242	14270	18654	13781

1909.—RECORD OF FLIES CAUGHT WEEK BY WEEK IN THREE DIVISIONS OF THE

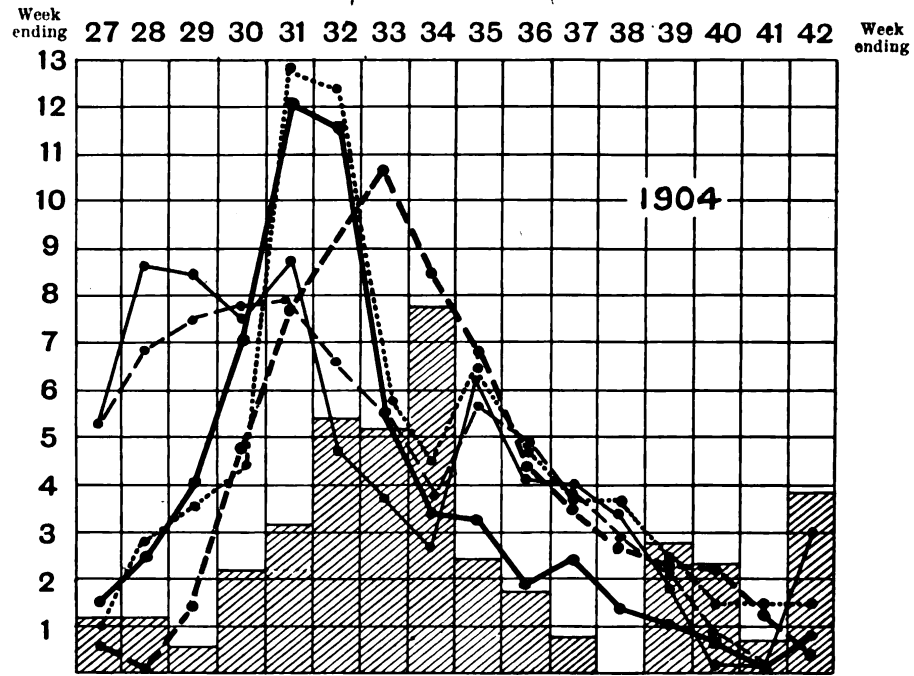
Divisions composed of		21	22	23	24	25	26	27	28	29
		May 29	June 5	June 12	June 19	June 26	July 3	July 10	July 17	July 24
Central Chorlton - on - Medlock ... Hulme ...	Flies (8 traps) ...	189	645	584	989	757	1487	1869	2489	3816
	Diarrhœa deaths...	—	—	—	1	—	1	—	1	—
	Fatal cases commencing ...	—	—	—	—	—	—	—	1	—
Ancoats ... St. George's ... Harpurhey ... Newton ...	Flies (12 traps) ...	403	1138	2123	4627	4102	6385	7382	10526	9700
	Diarrhœa deaths...	—	3	—	1	2	1	—	4	—
	Fatal cases commencing ...	1	1	1	2	1	—	3	—	5
Bradford ... Beswick ... Clayton ... Ardwick ... Openshaw ... West Gorton...	Flies (14 traps) ...	1953	2893	5550	6020	5926	8236	7947	7013	7597
	Diarrhœa deaths...	2	—	—	—	—	2	1	1	1
	Fatal cases commencing ...	2	—	—	1	1	1	—	4	2
Total number of flies ...		2545	4676	8257	11636	10785	16108	17198	22028	21113
Total deaths, diarrhœa ...		2	3	—	2	2	4	1	6	1
Cases commencing ...		3	1	1	3	2	1	3	5	7
Deaths according to date of onset ...		3	1	2	3	2	1	5	6	8
Mean temperature of air ...		55.8	53.9	51.0	56.1	55.2	57.7	58.6	58.5	58.3
Temperature at 4 ft. underground ..		49.6	51.1	52.0	52.4	53.4	54.0	54.9	55.9	56.6
Temperature, 1 ft. ...		54.0	54.5	53.2	55.4	56.7	55.8	59.3	59.3	59.7
Rainfall ...		1.150	0.320	0.060	0.285	1.400	0.390	0.780	0.795	0.320

Aug. 29	Sept. 5	Sept. 12	Sept. 19	Sept. 26	Oct. 3	Oct. 10	Oct. 17	Oct. 24	Oct. 31	Nov. 7	Nov. 14	Nov. 21
4	10	8	7	8	13	9	8	7	6	13	9	19
54	29	26	21	20	20	11	18	18	17	6	5	1
14	14	13	18	13	13	12	10	5	4	—	—	—
57.9	52.1	54.0	56.7	58.3	65.6	59.3	58.0	49.9	51.5	48.7	45.8	44.4
1.435	0.555	0.690	1.260	1.065	—	0.210	—	0.870	0.370	—	0.720	1.065
57.8	55.7	54.9	53.9	56.3	57.7	57.8	55.4	53.4	50.9	48.7	43.6	43.7
58.6	58.2	57.2	56.2	55.8	56.0	56.5	56.7	56.1	54.8	53.5	51.9	50.2
9436	7856	8832	6896	6439	6777	6635	5845	—	—	—	—	—

CITY, ALSO THE DEATHS FROM DIARRHOEA. STICK PAPERS USED. STATIONS, 34.

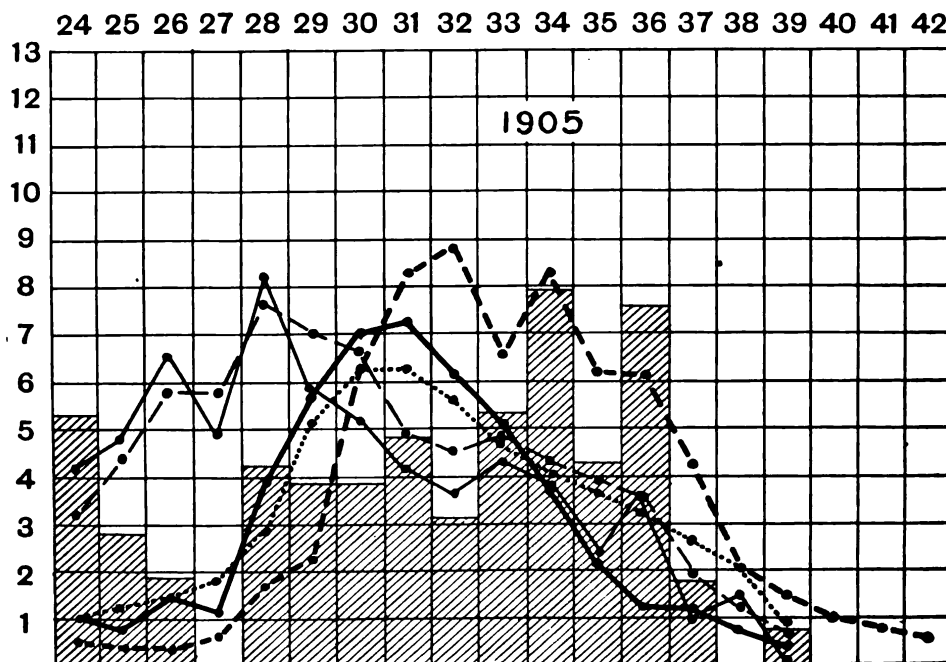
30	31	32	33	34	35	36	37	38	39	40	41	42	43	44
July 31	Aug. 7	Aug. 14	Aug. 21	Aug. 28	Sept. 4	Sept. 11	Sept. 18	Sept. 25	Oct. 2	Oct. 9	Oct. 16	Oct. 23	Oct. 30	Nov. 6
4336	6661	8388	9250	6658	5577	3986	4662	4604	—	—	—	—	—	—
—	2	—	2	3	6	3	4	6	2	6	—	3	1	—
—	3	1	3	5	3	2	2	4	4	—	1	—	1	2
7552	11093	14380	11078	9381	6863	4715	4247	3785	—	—	—	—	—	—
2	5	5	10	16	12	6	6	4	5	5	1	5	—	3
3	5	9	20	9	8	2	3	4	—	2	2	2	—	—
8874	10392	10327	10840	10160	12644	9439	7440	7157	—	—	—	—	—	—
3	4	7	5	6	6	5	5	4	1	1	2	1	—	—
6	4	4	5	6	2	1	2	3	2	1	—	—	—	—
20762	28146	33095	32168	26199	25084	18140	16349	15546	—	—	—	—	—	—
5	11	12	17	25	24	14	15	14	8	12	3	9	1	3
9	12	14	28	20	13	5	7	11	6	3	3	2	1	2
6	13	17	32	21	15	6	7	12	6	5	2	3	2	2
57.8	60.2	64.6	65.6	56.9	54.0	53.3	53.8	55.3	55.0	54.6	54.1	54.2	41.8	48.4
57.0	57.0	57.4	58.6	58.9	58.3	57.4	56.4	55.6	55.1	55.0	54.8	54.0	53.6	51.6
58.5	58.8	61.9	62.2	58.9	56.9	55.3	54.1	53.8	54.5	55.1	53.3	52.6	47.1	45.0
2.525	0.370	0.040	2.035	0.505	0.625	0.850	0.010	0.665	0.840	1.350	1.730	1.705	0.515	0.080

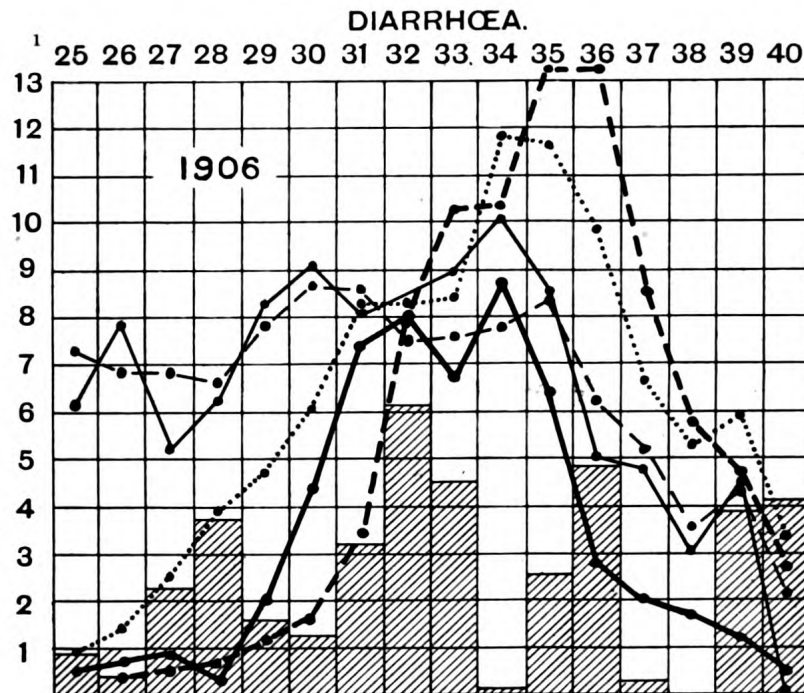
DIARRHŒA.



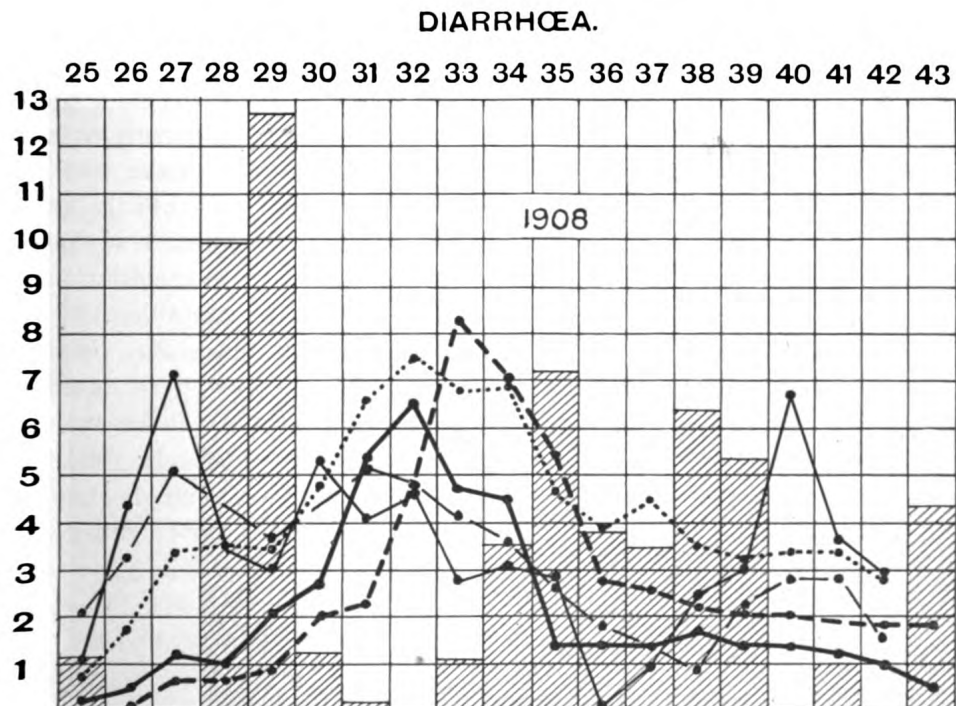
In this and the following four charts the curve — indicates fatal cases commencing
 indicates flies
 ,, atmospheric temperature — — — — — indicates deaths
 ,, 1 ft. temperature

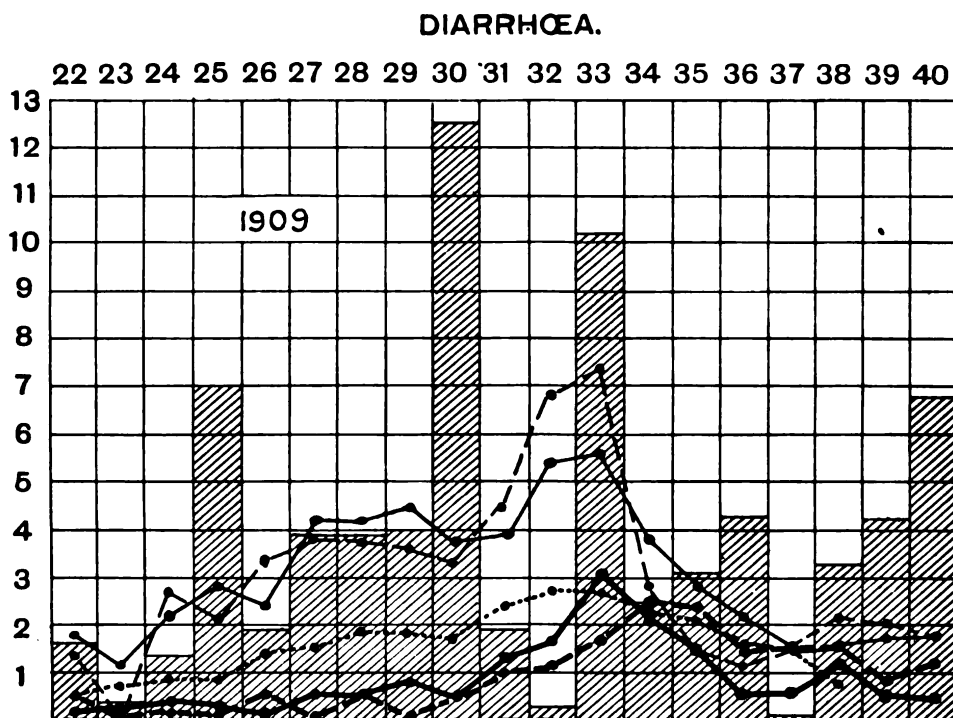
DIARRHŒA.





¹ The number of weeks at the head of this chart should be moved back by one place. Thus, 25 should be 26, and 40 should be 41.





The distribution of the deaths relative to the cases at the period of maximal fatality is accounted for. It is, however, evident that the interval of maximum attacks and deaths will not much exceed a week. It is probable that with the prolongation of illness the average severity of the attack decreases. This change in the severity of the cases is noted also in the annual report for 1904. We thus see a fairly swift evolution in character of the type of illness. Whether, however, this is owing to the invasion of more resistant individuals or to the dilution of infection in course of transmission we have no means of telling. It is possible, of course, that infection conveyed from without is less virulent than infective matter transferred directly from one person to another. There are, also, a number of cases at the very beginning of the upward movement of diarrhoeal deaths which have a long period. In 1904 the cases commencing show an upward movement in the fortnight, and in 1905 and 1906 in the third week preceding the upward movement of the diarrhoeal deaths.

RELATION OF CASES AND DEATHS TO THE TEMPERATURES.

We see at once that the course of diarrhoea deaths has no direct correspondence with the course of the atmospheric temperatures or with the temperature of the soil at a depth of 1 ft. On the other hand, a series of high atmospheric and 1 ft. temperatures at a particular period of the year is accompanied by a rush upward of the diarrhoeal fatality. The period at which this correspondence is observed coincides with the annual wave of flies. Fluctuations in these temperatures have but little effect on the course of the numbers of flies captured, on diarrhoeal cases, or on diarrhoeal fatality. Note the temperatures in the following weeks and their relation to diarrhoeal fatality: The thirty-fifth in 1904, thirty-second in 1905, twenty-seventh and thirty-second in 1906, twenty-eighth and twenty-ninth in 1908, weeks ending July 24 and 31 and week ending August 28 in 1909. Yet the relation of these temperatures to diarrhoea is undoubted.

It is otherwise with the temperature at a depth of 4 ft. Though the readings at a particular epoch may bear no constant relation to the course of diarrhoea as at the commencement of cases in 1904, 1905, and 1906, yet the course of the 4-ft. temperatures is parallel to that of the course of deaths both in its ascent and in its decline. It reaches its maximum in 1904 in the week preceding the maximum week of deaths, in 1905 in the week preceding, in 1906 in the same week, in 1908 in the same week, and in 1909 in the same week. There is therefore no close correspondence between the maximum 4-ft. temperature and the maximum fatality from diarrhoea, except in the general rise and fall, and Dr. Hamer's observations for 1907 show for that year a complete separation of periods.

With the course of the rainfall the curve of fatality shows no correspondence whatever, and it is evident that any effect exerted by rainfall must be indirect, and must be through temperature, effect on the transmitting agent, or in some other manner.

It is otherwise with the relation of cases and deaths to the number of flies captured week by week. Here the correspondence is close and intimate. The number of cases begins to increase when flies have reached a certain number, and continues to increase until the flies captured attain a maximum. The maximum of cases commencing in the years 1904 and 1905 is in the same week as the maximum of flies. The figures for the deaths are still more striking. The shape of

the curves at an interval of a week to a fortnight near the maximum point is practically identical in the two. The errors of the curves will be subjected to examination afterwards. But, even with their manifest and necessary defects, they show a degree of correspondence which creates a high degree of probability that flies are the transmitting agents in summer diarrhœa. In all the curves it will be seen that deaths diminish more rapidly than do flies in the middle part of the decline. For this there are two causes. In years of high diarrhœa-incidence the more susceptible and exposed infants have been killed off or rendered immune. In every year towards the close of the fly season the flies are attacked by *Empusa muscæ*, and are hindered by cold from leaving the house, so that they cease to act as transmitting agents.

At this point it will be convenient to recall certain features of the life of the house fly which have a bearing on the interpretation of the facts. The best accounts of the house fly and of the conditions of its increase which I have come across are to be found in a small book entitled "Our Household Insects," by Mr. E. A. Butler, published in 1893 by Longmans, Green, and Co.; in an article in *Public Health*, May, 1907, by Dr. Griffith, of Hove; and in Mr. Gordon Hewitt's monograph, part 3, to be found in the *Quarterly Journal of Microscopical Sciences*, December, 1909. Many interesting facts are to be found also in Dr. Hamer's contributions, entitled "Nuisance of Flies," reprinted from the annual reports of the Medical Officer of Health to the London County Council, 1907 and 1908. Reference should also be made to the publications of the Local Government Board.

I may say that early in these inquiries I felt the need of fuller information respecting the breeding of flies, and applied for information at the University. Mr. Hardy, of the Museum, gave me some information, *inter alia*, that there are at least two species of beetles which prey on the larvæ of flies, and that the shortest period of development of the house fly is about a week. At that time, however, I could not find accurate information on one of the points which I was hunting up—viz., the period between the deposit of eggs and the emergence of flies. The points which appear to throw light on our present inquiry are the following, taken from Mr. Gordon Hewitt's paper quoted above: *Musca* prefers to lay its eggs in horse manure, but will also use cow-dung. Gordon Hewitt was successful in rearing larvæ in horse manure, cow dung, fowl dung, and human excrement. Horse manure he found, as I also had done, swarming with larvæ. The larvæ will feed on paper, woollens, cotton garments, &c. They were also reared on decaying

vegetables and fruits, bread soaked in milk, and boiled egg. The larvæ were found in privy-middens, and also on a public tip among the warm ashes and clinker.

The shortest period from the laying of the egg to the development of the fly both Griffith and Gordon Hewitt found to be eight to nine days, at temperatures ranging from 95° F. to 71° F. A more common period is from twelve to twenty days. Absence of moisture leads to the production of a small fly. Cold also produces small and imperfect flies. Dryness also delayed development. Deficiency or unsuitability of food naturally impairs development.

In very hot weather the progeny of a fly may be laying eggs in about three weeks after its eggs have been laid. Usually, however, the period is longer than this. Flies are sexually mature in from a fortnight to three weeks.

The larvæ pass through three stages of varying durations. Under favourable conditions the three stages will be completed in less than a fortnight, and the development of the imago will take four or five days longer. The flies lay their eggs, in warm weather, on moist material of the character named. A female fly will lay four to six batches of eggs, each batch 120 to 150 in number.

Flies were shown by Griffith to breed in winter under suitable conditions, and in a good many situations odd ones may be found coming out of their recesses into warm rooms. There is little doubt that they find their way into hollow spaces and cavity walls, as in such situations, they lose a minimum of moisture owing to the stillness of the air surrounding them. Some years ago I observed a considerable number at midwinter in the basement of a common lodging-house, and I have been informed by two medical friends that they have seen odd flies as late as Christmas. Every here and there householders have the same experience. There is little or no doubt, in fact, that the seasons are connected by living flies and not by pupæ. Prolific as flies are, as we have seen, they are subject to numerous drawbacks in breeding: the lack of suitable temperatures, the lack of moisture, heavy rain, over-laying, predatory beetles, and so forth. The larvæ and the pupæ need quite different conditions. The larvæ must have warmth and moisture; the pupæ must have warmth and dryness. These conditions they evidently get *par excellence* in collections of horse manure. But it will also be evident that heavy rains falling on shallow collections will be unfavourable to the larvæ owing to the chilling produced by evaporation, and to the pupæ for the same reason. Such collections will be apt also to dry up too quickly.

Collections of horse manure roofed over will generally be too dry in warm, dry weather, and in various ways the brood may be prevented from developing. Only when the larvæ are deposited in a collection of horse manure, large but presenting fringes not liable to be heaped over, with access of moisture, but dry on the top, will the entire brood be likely to emerge. The same favourable conditions will also be found near the base of large tips. Only a small part of the eggs laid, we may be sure, come to fruition. Hence the numbers recorded, no doubt, represent the main course of the fly numbers, and appear to show that it should not be very difficult to arrest the whole process.

House flies are thirsty creatures, and must have access to moisture. It is doubtless this feature which makes them cluster round the face of the pedestrian in summer, and which causes them to beset the mouth and nose of the infant. They were observed in Manchester to cluster especially about the nose and mouth of infants suffering from diarrhœa. The same feature was observed in South Africa with regard to enteric fever. This I believe to be due to the development of a peculiar ether which I have noted in the breath of many cases of enteric fever. Driven by need of moisture, they betake themselves to cups of tea, bowls of milk, condensed milk, syrups, and beer. But they are also partial to sugar, and therefore visit sugar and breadcrumbs. Warmth also is agreeable to the musca. For all these reasons it prefers the kitchen to the parlour. The need for water is such that we must not accept the dictum that flies are driven indoors by rain. Possibly very heavy rain may have that effect.

It appears needless to trace the early course of the wave of flies, which probably finds its starting place about April in some covered fermenting collections of horse manure. As the ground and atmospheric temperatures rise, the number of swarms increases. I have already given reasons why the numbers do not reach greater dimensions early in the season. The reasons for the annual decline lie partly in the increase of cold, partly in the spread of the *Empusa muscæ*, which begins in July, becomes destructive in the latter part of August, and, as September advances, kills large numbers. Numbers also probably seek shelter. That *Empusa muscæ* is unable to prevent the growth of flies under favourable conditions is seen in the renewed swarms which appear in some seasons late in the autumn, under the influence of high temperatures; but it is also probable that the beetles which prey on the larvæ become more numerous as the season advances. It is possible that the flies which seek shelter are fairly numerous. I have seen spiders in

midwinter in very good condition, with not a fly about. Possibly mice do not disdain them. Mr. Butler states that there is no antagonism between flies and cockroaches: it is a point worth determining over again. It would be interesting to know what becomes of the dead flies; usually they are swept away, but those which are not seem to disappear very promptly.

It is evident that if flies are responsible for the ascent of the diarrhoeal curve, it requires a goodly number to start it on its upward course; in fact, we must imagine to ourselves an infant's food visited by a very large number of flies, some only of which convey infection from the excreta of a previous case. The quantity of infection in a given case will thus at first be small, and a number of slight cases will be produced, which scarcely attract notice; but as flies multiply, and cases multiply, the amount of infection conveyed to foods will increase. Meanwhile direct infection is going on, and probably the cases thus infected will, in general, be most fatal, those infected by flies becoming more fatal, but still less so than those infected by direct conveyance from person to person. As flies and cases continue to multiply, however, the fly-borne infection preponderates more and more, and we get a massive and fatal infection which quite overshadows the direct process. The average case thus produced remains, however, less fatal, and has a longer course than that owing to direct transmission.

With regard, now, to the manner in which we may suppose flies to carry infection. What we have to explain is why infants continue, in increasing numbers, to become infected without any evidence of exposure, at home or elsewhere. We may fairly assume that the freshly-emerged fly does not usually carry off from his birthplace any great amount of infection; he is spruce, young, active, and keeps himself clean. Setting out in search of food and drink, he betakes himself, amongst other places, to the most likely house he can find. Perhaps it is baking day, and there is a goodly assemblage of flies. The baby has diarrhoea, and they settle on its lips, or visit its napkin, which is tossed aside. Perhaps they light on some diarrhoeal excreta from an older child. The kitchen is cleaned up, and the flies now infected escape, in search, it may be, of moisture, and soon find themselves in another house where there is also an infant. Here they visit the various articles of food, and, if in sufficient numbers, leave a certain amount of infection behind them. The amount which they leave will depend, however, on how many centres of infection there are about; that is, on how many flies come from infected houses in which they have access to infected excreta, and

on how many have visited infected excreta, presumably for the purpose of depositing eggs. The chances will be very much against any one fly carrying infection, and the numbers must therefore be very large before infection is conveyed. That such is usually the case will be gathered from the particulars collected by Inspector Hewitt, and published in my annual reports for 1904 and 1905. It will be seen, too, that so long as the flies are healthy and active their power for mischief increases with their increasing numbers, as a steadily larger number will be visiting pail recesses and middens, and as the numbers of children attacked increases. This process is limited, as we have seen, by the increasing numbers who have recovered, and by the growing sluggishness of the flies.

There is no doubt at all about the extent to which flies move from house to house. This is gathered directly from examination of the records of individual stations, in which the numbers fluctuate remarkably. Equally little doubt is there about their visits to middens, and their return to fresh houses laden with the spoils of these places. If, however, we compare the incidence of diarrhœal mortality on different localities, although the influence of middens is unmistakable, we must conclude that all the phenomena of spread of the disease can take place in their absence. There is some presumption, at all events, that it is the transference of flies from house to house which is the chief factor in conveyance; the circumstances, however, forbid any exact conclusion. The infected flies may give the disease to the infant through its food, by direct contamination of its lips, or indirectly by way of some older person, and the observations published in my annual reports for 1904 and 1905 show that the latter mode of entrance of diarrhœa into a house is probably not infrequent. Nor, in this case, does the infection need to be carried into the house by flies; it may come in syrup, milk, bread, or fruit which they have visited in some shop.

It does not follow because older persons have not had a well-marked attack of diarrhœa that they have not been infected and so introduced the disease. The remarkable series of cases published in the *Manchester Annual Reports* for 1905, and cases given in other reports, show that enteric fever has been contracted and recovered from without almost any ill-health. No doubt precisely the same is true of summer diarrhœa. Now we have proved that slight cases of overlooked enteric are, for purposes of infection, far the most potent source of direct infection. Nevertheless, direct or contact infection is a small factor in the causation of the annual wave, though not unimportant in determining its

magnitude. Nor is the remarkable correspondence between the curves of diarrhoeal fatality and the curve of flies confined to our observations in Manchester. Dr. Hamer's curves of mortality for 1907 and 1908 correspond in a remarkable manner to the curve of flies which he has given for the stations surrounding stable centres. Now it is precisely from these centres that we should get swarms of young, active migratory flies. How far do these migrations extend? From observations on marked flies carried out at Monsall Hospital Dr. Miles B. Arnold has shown that they can travel a distance of 190 yards. It is not to be supposed, however, that they do, in fact, in the heart of towns often take such flights; nevertheless, it is a valuable proof of their power and inclination to travel a considerable distance. Dr. Hamer has thrown out the suggestion that the numbers required in different years to cause infection may be different, depending on the initial centres of infection, as it must do. That appears highly probable, especially at the commencement of the outbreak; but the variation is likely to be least where the number of endemic centres is large at all times, as is probably the case in Manchester, and it is not likely to have much effect towards the summit of the curve if diarrhoea reaches any great intensity. The migrations of flies, however, may be quickened by warm, dry weather, and the number of infected flies reaching a given house will certainly be dependent on the total number of flies and on the centres of infection within reach. Hence the fluctuations in diarrhoeal mortality in sparsely peopled outlying districts, and in districts with a low average diarrhoea-incidence, are likely to be much greater than in a densely inhabited district with many endemic centres. What determines the number of endemic centres at the commencement of the season we do not yet know, though we can assume it to be a function of the average incidence. It is greatest amongst the poorest classes, who also possess in general the least well-constructed and the least well-kept houses. On reference to the table of district mortalities we do, in fact, find that fluctuations are greatest in the better-off, least densely peopled districts. It is difficult to see how this is to be explained on any theory of soil influence. If, now, the number of flies required to carry infection varies with weather, being least in warm, dry sunny weather, largest in cold, wet weather, we must not expect too close a correspondence between flies caught and fatal cases commencing. The actual degree of correspondence would seem to show that the adverse factors named do not operate as much as we should expect.

The question of the number of flies required to produce an effect on the diarrhœa curve is one of considerable moment, if we assume, as I now propose to do, that the relation is one of cause and effect, a matter to which I will hereafter return. Referring to the records, we find that the number of cases begins to increase in the following weeks: In 1904, in the twenty-seventh, in which the number of flies captured was 1,498 in 11 beer-traps; in 1905, in the twenty-fourth, with 1,527 in 14 beer-traps; in 1906, in the twenty-fifth, with 1,984 flies in 17 beer-traps. The corresponding number of flies per 10 beer-traps is thus shown:—

Year			Week			No. of flies
1904	27	1,362
1905	24	1,090
1906	25	1,170

As regards the end of the curve, in 1904 the last considerable number of cases commencing is in the fortieth week, when the number of flies captured was 2,398; while in 1905 the corresponding week was the thirty-eighth, and the number of flies 2,978. In 1906 the curve of cases does not represent the curve of deaths; if, however, we measure back three weeks from the end of the curve of deaths, we find that the number of flies was 3,349. The number of flies, therefore, at the end of the period was greater than at the beginning. It is doubtful, however, whether these flies at the end of the period had much, if anything, to do with the continuance of the curve of deaths, which, for reasons already given, is more probably dependent on direct infection at this point.

It may here be pointed out that we must not attach too precise a value to numerical comparison of deaths and flies at different points of the curves. Undoubtedly the number of flies effective to produce an effect on the diarrhœal curve at the outset must be much more potent for purposes of infection than the number at the height of the fly season. Many of these are engaged in reproduction, and are not available for transmission of disease; many others are already invaded by *empusa* and will not be available either for reproduction or for carrying infection. On the other hand, the centres of infection have multiplied to such an extent that the fresh broods coming into play can effect much more mischief than could a similar number of flies at the commencement of the curve. The relations are too complex for the establishment of numerical relations. The numbers are available only for comparison near the points of the curves to which they refer.

RAINFALL AND FLIES.

We may now consider the relations of rainfall and temperature to flies. These are not now under consideration in their bearing on infection, but it appears desirable to see how they affect the curve of flies, bearing in mind that this curve needs correction and that in any attempt to reconstruct the course of the fly curves we should get valuable aid from such a study. We may begin with the relation of rainfall to flies as being the least involved.

The effect of rainfall on flies will be gathered from the summary of fly observations. In 1904 there is a fairly heavy rainfall in the thirty-second, thirty-third, and thirty-fourth weeks. Coincidentally with this there is a fall in the number of flies and of deaths, and also in the various temperatures. Nevertheless with diminished rainfall in the thirty-fifth week, and rise in the mean temperature of the atmosphere, there is increase in the number of flies captured and arrest in the decline of cases of fatal diarrhœa. Recovery is thus rapid though imperfect. But the diminution of flies and cases in the three weeks is very marked. In 1905 there is over an inch of rainfall in the twenty-fourth week. This does not prevent considerable rise of all temperatures. Rainfall of nearly an inch in the thirty-first week accompanies the maximum of flies and of fatal diarrhœa commencing. There is, however, fall of the atmospheric and 1-ft. temperatures, the latter marked. Rainfalls of over an inch in the thirty-third and thirty-fourth weeks are accompanied by rise in the atmospheric and 1-ft. temperatures, but accompany a fall in the number of flies and of fatal cases commencing. Rainfall of $1\frac{1}{2}$ in. in the thirty-sixth week is accompanied by rise of atmospheric temperature with fall of the 1-ft. temperature. There is not marked fall in the number of flies, though there is in the number of cases, in the fortnight following. In 1906, in the thirty-third and thirty-fourth weeks, rainfall of about an inch is accompanied by some fall of atmospheric temperature, which, however, rises sharply in the second week. There is stasis in the first week in the number of flies and in the number of fatal cases commencing, and rainfall of about an inch in the thirty-sixth week is accompanied by fall in the number of flies, number of fatal cases commencing, atmospheric and 1-ft. temperatures. In 1908 heavy rainfall in the twenty-eighth and twenty-ninth weeks is accompanied by diminution in the number of flies captured in the second of these weeks, and by falls in the atmospheric and 1-ft. temperatures. The decline of the 1-ft. temperature is most noticeable in the twenty-ninth week, and it is not until the

thirtieth week that stasis of the 4-ft. temperature is evident. It is observable that the upward movement of flies remains partially affected in the thirtieth week. In the thirty-fifth week rainfall of about $1\frac{1}{2}$ in. is accompanied by marked diminution in flies and of the 1-ft. temperature. In 1909 rainfall of 1·4 in. in the week ending June 26 is accompanied by fall of atmospheric temperature and stasis of 1-ft. temperature, also by stasis in production of flies. Rainfall of 2 in. in the week ending August 21 is accompanied by stasis in the number of flies, and in deaths a fortnight afterwards. The mean temperature of the air rises, as do the ground temperatures in the same week.

Thus heavy rainfalls tend to lower the temperature of the surface, but have less effect on the atmospheric temperature, which may rise in spite of them. They produce a greater effect on flies than they do on temperature, and this effect on flies is reflected in the number of fatal cases commencing, and in the number of deaths in the week but one following. This is not a quite accurate statement for the end of the curve. But at this period rain will have less effect in reducing the number of flies captured than on deaths, since it is on the production of young active flies that their chief influence is exerted, and the number of these is not exhibited in the numbers now captured. The influence of rainfall when excessive is thus not exerted entirely through its effect on the general temperature. It has a marked influence on flies and deaths apart from this. It is probable, however, that the influence is really on caloric in the first instance. Smaller collections of refuse, privy-middens, and small shallow collections of horse manure get saturated and unfavourable to the pupal stage. The numerous points which they present permit of rapid evaporation exceeding that of surfaces in general, with consequent chilling of the imago. Moreover the surface temperature is different from that of the atmosphere or that at a depth of 1 ft., and is probably lower than either, owing to evaporation. There can be no doubt that heavy rainfall exerts on the whole a disastrous influence on the production of flies. This does not occur at rainfalls of 0·8 in. or under, which appear to have the reverse effect, at all events so long as the atmospheric temperature is rising.

In this nice balance more or less of rainfall over the quarter cannot much matter. In fact, in Manchester no sustained correspondence can be made out between mean rainfall in the third quarter and the number of deaths from diarrhœa. The years of highest rainfall—viz., 1891, 1892, 1893, 1895, and 1903—have all been years of fairly high diarrhœal fatality; 1907, the year of lowest diarrhœal fatality, was not a year of

exceptionally low rainfall. Even heavy rainfall, it will be seen, does not necessarily exert any unfavourable influence on the development of flies or the extension of diarrhoea. Its doing so will depend entirely whether it is able to lower the surface temperatures below those which are favourable to the development of the larvæ or the escape of the imago. If it fail to effect this, its influence will probably be in the opposite direction, owing to the great need of moisture for the development of the larvæ and, one may add, for the health of the flies.

It appears desirable that we should now consider how the continuous course of the 4-ft. temperature arises, and how it comes to be related to the continuous course of the fly curve and of the diarrhoeal curves.

THE COURSE AND INTERACTION OF TEMPERATURES.

From the data for any year it will be seen that the atmospheric temperatures ascend much more rapidly during the summer than do those at the depth of 1 ft., and that these in turn ascend and descend much more rapidly than do those at a depth of 4 ft. The fluctuations of the atmospheric temperatures, however, are generally reflected in the mean temperatures for the same week at a depth of 1 ft., though much reduced in amount. At a depth of 4 ft. the oscillating upward and downward movement of temperatures disappears and is replaced by a fairly steady upward and downward curve. Why is this? We may divide the soil into layers of 1 ft. in thickness, the 1-ft. thermometer being placed at the junction of the fourth and second, the 4-ft. thermometer at the junction of the fourth and fifth. In the long bright days of June radiant heat is absorbed by the surface layer of the soil, and by numerous other surfaces, which in their turn warm the air in contact with them by convection. Part of the radiant heat is absorbed and causes the temperature of the surface layer to rise, according to its specific heat and conductivity. Only a portion of the heat absorbed reaches in, say, two days, the second layer of soil, those portions being lost which are radiated off from the surface, or used up in raising the temperature of the layer, or used to a small extent in supplying chemical energy and energy of growth, or expended in evaporation at the surface. The inward flow of heat is accelerated or retarded by the movements of heat at the surface, which are therefore reproduced on a smaller scale. Exactly the same applies to the flow from the second to the third sheet, from the third to the fourth, and so on; the oscillations in the flow inwards or outwards getting rapidly reduced, until at a

depth of 4 ft. the movement is practically continuous. At each successive layer, the first effect of inward flowing heat is to raise the temperature of the layer, a circumstance which rapidly reduces the size of the oscillations. According to Copeman, in Sir Shirley Murphy's "Treatise on Public Health," the annual movement of heat reaches a depth of 60 ft., the movement becoming slow and uniform when we reach some depth. The 4-ft. thermometric readings therefore constitute a fairly uniform register of the excess of heat received at the surface over heat lost by the first 4 ft. of earth from February to August, and of loss over receipt from August to February. Any growth of bacteria, however, must partake of the fluctuations observed at or near the surface, and must be subject to the effects of rain in chills as well as arrest of movement from the surface. The growths and their dispersion would therefore tend to partake of the changes of temperature and dryness at the surface. This opinion is strongly supported by the known rapidity of production of cultures of bacteria. They would almost certainly suffer acutely or flourish exceedingly, the more so that the periods of favourable and unfavourable surface conditions are often of considerable duration. There is, however, no sign of such vicissitudes in the progressive and decided curve of diarrhœal fatality.

It is easy to see, from study of the course of temperatures, that the wave of inflowing heat takes rather over a week to reach the depth of 4 ft. The course of the 4-ft. temperature corresponds generally, therefore, to the excess of heat entering over heat leaving the surface in the week before. But it is a summation, and it does not even follow when the 4-ft. temperature continues to ascend that there has been any heat added in the week previous. There may have been defect, the difference being made good from the absorbed heat stored up in the four top feet of ground in the weeks before. Nevertheless, the ascent of the 4-ft. temperature indicates in general continued gain of heat at the surface over loss a week before, the amount thus stored up flowing back if need be to make good loss from lowering of the surface temperatures.

Now, Dr. Griffith and Mr. Gordon Hewitt both show the need of heat for the development of the larvæ. How is the inflow of heat indicated by the 4-ft. temperatures related to flies? Imagine a collection of horse manure in an open midden rising to a height of 3 ft. or 4 ft. If the bottom of the receptacle is some 2 ft. below the surface, the manure takes at the sides the mean temperature of the surrounding earth, and the larvæ, which prefer the bottom of the heap, are deprived of heat if the surrounding soil is at a comparatively low temperature. With a

large heap of fermenting manure in the receptacle, sufficient heat will usually be generated to raise the temperature of the bottom layer. But the temperature of this layer will depend on the difference in level between its temperature and that of the surrounding ground. Now, so long as the surface layer is receiving more heat than it is losing, its temperature rises, and the heat lost from the manure heap is, *pro tanto*, reduced. That is to say, the larvæ feeding at the bottom of the heap are under steadily improving conditions so long as the 4-ft. temperature is rising. A small heap of manure is much more dependent for its heat on the ground than a large one, and is also much more dependent as regards the starting of those internal processes of fermentation which raise its temperature. The same applies to middens.

If from any cause, such as rainfall, the temperature of the manure heap is lowered, inflow of heat takes place from the soil around and beneath. The temperature of the soil at various depths is thus of considerable importance to the growing larvæ, and it is this influence which the 4-ft. temperature integrates and registers. The pupæ, on the other hand, need a warm, dry atmosphere. Much rain, by causing evaporation and chilling, is hostile to the emergence of the fly. Now, the state of the atmosphere may not be determined by the condition of the surface, though it generally is. To some extent there is independence in the conditions determining a favourable surface and a favourable interior of the manure heaps in which flies are generated. In general, however, sunny, warm weather is favourable to both. Thus at the commencement of the season *musca* probably starts to increase in a few isolated, considerable, fermenting manure heaps, and large tips. As heat flows into the ground more manure heaps start fermenting, and by now there are more *muscæ* laying eggs; multiplication increases. Towards the height of the season every heap of refuse and excrement receives in a warm year sufficient heat from the soil to support the larvæ. In comparatively cold years it is the smaller heaps of refuse and middens which are cut off from producing. Moreover, the stages of evolution are longer, and a larger number of flies perish from being overlaid. This, however, is less liable to happen in middens and tips than in heaps of horse manure. These considerations explain the marked fluctuations in death-rate from diarrhoea observed in the districts served by privy-middens. *Homalomyia*, as we have seen, can apparently complete its evolution at a lower temperature than *musca*. It is probable, therefore, that in comparatively cold years *homalomyia* will be found in proportions to *musca* relatively high.

What is it that determines the fall in the curve of flies ? As we have seen, great numbers are destroyed by empusa. The fall coincides, however, with the period at which heat begins to be lost from the ground, and the loss is, of course, most rapid at the surface. We are thus, again, reminded of the great importance to the larvæ of a high temperature in the upper layer of the soil. There is still much laying of eggs, but the conditions of ground and atmosphere become, as a rule, steadily less favourable. If a spell of warm weather sets in again, however, flies increase, and diarrhœal fatality may also increase. For this purpose, however, the increase of flies must be considerable, as we are now dependent for carriage of infection on the fresh brood, the older flies having become comparatively inactive, and there may also be comparatively few infants left unattacked. Hence the increase of diarrhœa is usually small compared with the rise in temperatures. Flies, then, in their development and reproduction, respond to all the influences which affect the 4-ft. temperature : rain, sunshine, high absorption of surface heat, warmth and dryness of the atmosphere, and sum up in their annual wave these influences, though not just as the 4-ft. temperature does. Both correspond to the course of gain and loss of heat by the surface.

Flies stand, however, in a more intimate relation to deaths from diarrhœa than do the 4 ft. temperatures. The reaction to their increase probably takes place within two or three days, judging from the daily records. In all cases their increase precedes that of deaths by a week to a fortnight, except where we may confidently reckon on an error in the numbers captured, regarded as representing the average numbers. Yet on this point some caution is necessary ; it is easy for such an error to occur. But it is also quite possible that we might have a great increase of flies round a nest of diarrhœa with a general decrease in flies, but with increase in diarrhœa. In other words, it is necessary to bear in mind that the curve of contaminated flies is not necessarily coincident with the curve of all flies. There is, of course, this great difference between flies and the temperature at a depth of 4 ft.—that the former can directly influence the course of diarrhœa, the latter cannot.

The second line of reasoning by which we are brought to the conclusion that the annual rise of diarrhœa is caused by the annual uprush of flies consists, therefore, in the establishment of a close correspondence between the number of flies captured in any one week and the number of cases of diarrhœa occurring in the same week, or the number of deaths occurring about ten days subsequently, coupled with the absence of any other satisfactory cause. The first line of

reasoning was deduced from a study of the distribution of diarrhoeal mortality in the sanitary districts of the city, based on the table given at p. 135. The behaviour of diarrhoea in this table is the well-known behaviour of an infectious disease. The districts change their order of diarrhoeal mortality from one year to another, a circumstance which indicates a change in the proportion of susceptible material, or of centres of infection, or of carrying agents, or of all three. We have seen, however, that in years of comparatively low temperature in the third quarter, middens will probably take a minor part in the production of flies. Now, if flies are the cause of the summer rise of diarrhoea, there should be a comparatively small fatality in midden-privy districts in colder seasons. The mean temperature of the third quarter is, however not the best measure of the heat available for the production of diarrhoea, which may be very unevenly distributed. The best measure, probably, is the rate of mortality from diarrhoea during the quarter. The necessary comparisons, however, cannot be made in the last three years, during which period there has been a rapid reduction in the number of middens. During the period covered by this table there have been but few years of sustained low temperature. The most convenient will be 1896, 1902, and 1903. The total series of mean temperatures and diarrhoeal death-rates in the third quarter is as follows :—

Year		Death-rate per thousand living		Mean temperature third quarter. Degrees Fahr.		Rainfall third quarter Inches
1896	...	2·93	...	58·5	...	9·7
1897	...	6·01	...	58·9	...	9·7
1898	...	6·00	...	60·1	...	6·1
1899	...	6·96	...	60·8	...	7·7
1900	...	4·14	...	60·3	...	9·6
1901	...	6·33	...	61·9	...	6·5
1902	...	0·88	...	57·6	...	5·9
1903	...	2·19	...	57·8	...	12·3
1904	...	4·48	...	60·2	...	6·9
1905	...	3·89	...	58·9	...	9·4
1906	...	4·91	...	60·8	...	6·2
1907	...	0·45	...	58·5	...	7·8
1908	...	2·61	...	59·2	...	10·7

Now, the districts which up to recently have been predominantly served by midden-prives, and not by either water-closets or pail-closets, are Bradford, West Gorton, Clayton and Openshaw, while the pail-closet districts were St. George's, Ancoats, Central, and in large measure Hulme. If, therefore, flies are largely concerned in the spread of diarrhoea, we should expect that the diarrhoeal mortality would rise in

privy-midden districts in seasons of high temperature and low rainfall, and would fall under the reverse conditions. As we have seen, the effect of rainfall on the production of flies will be specially felt by small collections of manure and by middens. We find accordingly that in the season of highest diarrhœal mortality—viz., 1899—West Gorton and Bradford take the premier position, while in the next highest—1901—Clayton takes the first place. In 1898, again, Bradford, West Gorton and Clayton come before Ancoats and St. George's. In 1902 and 1903 Bradford and Clayton fall below the Central districts. Not so West Gorton, which in 1902 stands highest of all. The privy-midden districts in 1902, again, do not fall so low compared with their own mean as do the Central districts, while in 1903 they fall much lower. That has to do, no doubt, with the high rainfall in 1903 as compared with that in 1902. In 1896 their death-rates from diarrhœa are comparatively low in regard to their own means, except Clayton. Thus, while there is a general tendency in the direction which theory would indicate, the facts lend only a general support to the theory. There are clearly other and baffling factors at work. Such factors are the establishment of immunity in persons above the age of one year, the varying facilities for the production of fly swarms, and the varying amounts of infection available as a nucleus. All that we can assert is that the table indicates an infectious disease subject to local factors of the kind indicated, and is not consistent with the view of a general growth of bacteria in or on the soil. It may be pointed out that the quarterly seasonal factors in 1897 and 1908 are similar, with very different mortalities. This may be supposed to be due to the abolition of middens, but it is rather too soon to come to a positive conclusion, although the figures for Bradford, Clayton, West Gorton and Openshaw would seem to justify this conclusion. We have not gained so much from this line of argument as we might have hoped, but nevertheless the table forms an important part of our reasoning as regards infection.

Another promising line of investigation is to take different districts of sufficient size, plant in them a sufficient number of house-fly stations, and compare the incidence of diarrhœa deaths or cases commencing with the number of flies captured. If the course of fatality in the areas chosen in different parts of Manchester varies, then, if house flies are carrying the disease, the numbers of house flies captured in those areas will vary to correspond. That such differences do occur in the course of fatality in the main divisions of the city is clearly shown by the table just considered. Prior to 1909 I was not able to try this plan, and,

unfortunately, owing to the small number of flies and the low diarrhoeal fatality in this year, the observations are of little value. In 1906, however, the number of deaths was much larger, and the difference in their distribution in different districts was very striking; unfortunately, the number of fly stations in any one district was small, and no decisive conclusions could be drawn, yet the results, as far as they go, support the view that the disease is propagated by flies. They may be stated thus:—

FATAL CASES OF DIARRHŒA.

District	Beginning in		No. of fly stations	Maximum number of flies week ending
	June and July	Aug. and Sept.		
Ancoats ...	49	29	3	July 29, one station August 19, two stations
Newton ...	22	24	0	—
Bradford ...	15	14	1	July 29
Ardwick ...	17	13	1	July 29 and August 12 equal
Chorlton-on-Medlock	18	17	1	July 22 and 29, again an increase August 12 and 19
Central ...	14	27	1	August 12
St. George's ...	34	51	1	August 12
Cheetham ...	3	15	0	August 19
Beswick ...	4	16	1	August 12
Clayton ...	2	11	1	August 5
Openshaw ...	8	15	1	August 12
West Gorton ...	11	28	1	July 29 and August 19
Hulme ¹ ...	18	26	1	August 19

It will be conceded that these data, scanty and defective as they are, are very suggestive, the more so as Ancoats, Newton, Bradford, Ardwick, and Chorlton-on-Medlock are contiguous districts.

Insufficient as are the data for 1909, they have been given in the collected records, to which I must refer back. In this year stick papers were used, and each stick paper captured more than double the number of flies caught by beer-traps. I neither like the method nor am I sure that the results are quite comparable with those previously obtained. It is possible that homalomyia occurs in much larger proportion in beer-traps than on stick papers, and it is certain that homalomyia is more likely to visit middens to lay its eggs. Be that as it may, we may divide the total numbers of flies at least by six to get figures comparable with those for 1904 and 1905; probably the divisor should be greater. This being premised, it may be stated that three large areas were chosen. In the

¹ The number of flies in this district, however, in the weeks preceding greatly exceeded those in the weeks following.

first were planted eight stations ; in the second, twelve ; in the third, thirteen. The number of flies in each is insufficient to produce decided effects, the curve of deaths being irregular and indefinite in the first. In the second, however, both curves towards the apex are definite and very similar. Generally speaking, the apices of both curves precede that in the first area by a week. It is certainly interesting to find that the only area which gives a definite peak of flies also gives a definite peak of deaths. In the third area the number of flies is small and the apex not well marked ; there is a general rise of deaths corresponding, the summit of which is level and extends over six weeks. The greatest incidence of flies in the third area corresponds to that in the second, but is insufficient to produce a marked curve. The total curves give the usual results, the curve of flies preceding that of diarrhœa deaths by a fortnight. Indeed, the correspondence in the total curves is very striking once these are established. Perhaps the energies of the flies were used up at the commencement of the season in establishing centres of infection.

On the whole, these figures, which go for little, sustain the connexion between flies and fatal diarrhœa.

ON CORRECTION OF THE CURVE OF FLIES.

The enumeration of flies suffers from the small number of stations, and is liable to suffer if these are injudiciously selected. We may therefore occasionally find misfits in the curve of flies. We may, I think, apply corrections by observing the following considerations. Notwithstanding that the number of stations is small, it is probable that the numbers of stations in which there is increase or decrease in any week as compared with the week preceding will show the trend of the fly movement when the number of flies captured has gone astray. Then, moreover, there are certain broad features of the curves : The curve of cases begins to move a fortnight or three weeks before the curve of deaths. The intervals are : 1904, a fortnight ; 1905, three weeks ; 1906, a fortnight ; 1908, a fortnight. The greatest number of fatal cases begins in 1904, in the same week as flies reach a maximum. This is also the case in 1905. In 1908 and 1909 the maximum of cases commencing occurs in the week following the maximum week of flies. At the same time, any stasis in flies at this period is attended with diminution of cases. That is probably dependent largely on the influence of the fly fungus. An upward movement means

the coming of a large number of young and active flies. There is thus a presumption that in the higher parts of the curve and up to its summit, in years of moderate and considerable intensity, the number of cases commencing corresponds to the number of flies, so long as that number is rising. The maximum number of deaths follows the maximum number of flies, being sometimes in the week following, sometimes in the week but one following, the interval between deaths and cases being somewhat the shorter. In 1904 the interval is a fortnight, in 1905 a week, and here the two-weekly plateau in both is very striking. In 1906 the interval is a week, and here again the two-weekly plateau is striking. *Cæteris paribus*, up to near the apex, the same number of flies should produce more cases as the weeks advance, owing to the previous increase in infective matter. This is well marked in 1906. In 1908 the interval is a week. In 1909 the small two-weekly maximum follows the small fly maximum at an interval of a fortnight, the same correspondence at the apex being noted. It has already been explained that the actual interval between cases and deaths is somewhat over a week but less than a fortnight, and in the case of flies somewhat greater; but if regard be had to the fact that the maximum of production will fall in different parts of any given week, the uncertainty of the interval becomes matter of course. Moreover, the average interval probably varies from year to year.

Defective as are the numbers of cases with definite dates of commencement, and imperfectly, therefore, as they represent the curve of deaths, we may obtain some light on the distribution of flies from the arrangement of fatal cases commencing in different weeks. These indicate, as do the deaths, that the actual maximum of flies was in the week preceding the maximum of deaths, while the number of flies in the weeks preceding and following this were probably equal.

But there are also other points by which the movements of flies may be fixed. The 4-ft. temperature attains its maximum in the weeks :—

Year	Preceding or following maximum of cases		Preceding or following maximum of deaths		Preceding or following maximum of flies	
1904	...	Week following	Week preceding	Week following
1905	...	Same week	Week preceding	Same week
1906	...	Week but one following	Same week	Week but one following
1908	...	Week following	Same week	Week following
1909	...	Week but one following	Same week	Week but one following

The movements of the 4-ft. temperature do not therefore correspond closely with those of the curves of deaths, flies, or cases; still less do they do so at the commencement of the curves. These movements therefore cannot be used to correct the curve of flies. We may say, generally, that the number of flies at any period of a curve depends on the number of eggs laid three or four weeks before at the commencement of a curve, or a fortnight or so before in a warm season near the apex of the fly curve. It depends on the presence at that period of bright, sunny weather. It depends, also, on the surface temperature, high temperatures favouring the development of pupæ, especially in sunny, dry weather. It depends on the temperature of the surface of the ground a week before, which in its turn influences the development of larvæ. It depends also on the rainfall, much rainfall lowering the surface temperature and hindering the emergence of flies. The effect of rainfall is, however, much influenced by the atmospheric and ground temperatures, since with moderate rainfall and high temperatures there is increase of fermentation in manure heaps and accelerated development of larvæ.

From a careful consideration of the weekly data we may therefore form a very fair estimate of the probable development of flies. We may, by means of these data, construct fly curves even without reference to the number of deaths from diarrhœa, and, though a rough correspondence is thus obtained, it is by no means so close as that actually observed even with our deficient observations. Our chief object being, however, to correct the curves, we may use all the data available for this purpose, remembering that in the latter part of the fly curve it is impossible to determine how much is young, active carrying flies and how much fungus infested and disabled flies.

We may now apply these considerations to successive years:—

1904.—Applying first the numerical test to the number of flies captured in houses at the apex of the curve, we find that in the thirty-second week there were more stations showing an increase over the thirty-first week than there were showing a decrease. It may be assumed, therefore, that the figures in the thirty-first and thirty-second weeks represent substantially the numbers of flies at this point. As we have seen, the greatest number of deaths is probably about the ninth or tenth day after the greatest number of flies. If now we add the number of deaths occurring in the weeks and fortnights following the maximum number of flies, we get figures which represent nearly this point of time, and the features of the fly curve at this point are reproduced. This is

interesting as bearing on the number of flies found in the houses in the thirty-second week. We find throughout that heavy rain does not increase the number of flies in houses. Very often the reverse is the case. Flies, therefore, are not to any great extent driven into houses by rain. But heavy rain may keep a section of them indoors, those, namely, which have come in to feed, and which are not driven out by thirst—a very powerful factor in determining the movements of the house fly. Now, the weather was not very favourable to the production of flies in the thirty-second week. The atmospheric temperature fell considerably, and there was a fairly heavy rainfall. On the other hand, there had been an enormous number of eggs laid during each of the four preceding weeks, so that the impulse of the fly to escape from the pupa was only partially checked. Notwithstanding the unfavourable conditions, a large proportion of the flies captured would still be freshly generated flies which had escaped direct from horse manure into the houses. If we accept the figures, also, the movement of these flies from one house to another was only partially restricted by rain. It would seem from the data given in these tables that an atmospheric mean temperature of 59° F. and upwards is not unfavourable to the production of flies, although the higher the temperature the more favourable it is. This temperature, however, is too low for growing larvæ, which, however, at the beginning of the season receive the artificial heat of large fermenting heaps of horse manure. A great increase in the atmospheric and surface temperatures occurs in the twenty-eighth week and onwards for four weeks, and a sudden increase in flies occurs in the same week. It will be evident that a considerable amount of laying of eggs in favourable spots has occurred prior to this period, especially in the twenty-fifth and twenty-sixth weeks. It is not, however, till the twenty-eighth week that the temperatures are very favourable for the laying of eggs. An enormous deposit of eggs takes place, and a fortnight afterwards occurs the great uprush of flies. In spite, however, of the large number of eggs laid in the thirtieth and thirty-first weeks, there is a great decrease of flies in the thirty-third week under the unfavourable conditions then prevailing. Hence a low surface temperature is able to arrest the production of an enormous number of larvæ. With the advent of high atmospheric and surface temperatures in the thirty-fifth week, we get a second increase of flies and a check in the diminution of fatal diarrhoea cases. This appears to show that as a result of the low temperatures of the air and surface in the two weeks preceding, the development of larvæ had

been delayed, but was able to attain completion under more favourable conditions.

1905.—In this year the conditions are favourable to laying of eggs in the twenty-fifth, twenty-sixth, twenty-seventh, twenty-eighth, and twenty-ninth weeks; we therefore expect, if the conditions are favourable, a marked increase of flies in the twenty-eighth, twenty-ninth, thirtieth, thirty-first, and thirty-second weeks. There is, however, a diminution in the thirty-second week corresponding to less favourable atmospheric conditions; nor is improvement manifest in the thirty-third week, despite favourable atmospheric and surface conditions. This is due, no doubt, to the high rainfall causing chilling of the smaller heaps of material; the curves pursue a normal course.

1906.—The conditions for laying of eggs became favourable in the twenty-fourth and twenty-fifth weeks. We expect a considerable increase of flies in the twenty-seventh and twenty-eighth weeks, which occurs notwithstanding unfavourable air and surface temperatures in the earlier periods. The rainfall is, however, not unfavourable. The conditions for laying eggs are favourable in the twenty-seventh week, and we expect a considerable increase of flies in the thirtieth week, the conditions being favourable, which they are. The subsequent course of the flies is such as we should expect.

It seems unnecessary to follow the course of flies, and therefore of deaths, into further years.

We may sum up the results of this analysis as follows: Summer diarrhœa is an infectious illness. This is shown by the course of the annual wave, by the manner of its incidence on the different sanitary districts of Manchester, and by the history of individual cases. The health of infants prior to attack—in other words, the social condition—has much to do with the fatality. The summer wave is not due to dust, nor is it conditioned by any growth of bacteria in or on the soil. There is nothing to support the view that the infective organisms are of animal origin, and the connexion between privy-middens and diarrhœa goes far to prove the contrary. The disease becomes more fatal only after house flies have been prevalent for some time, and its fatality rises as their numbers increase and falls as they fall. The correspondence of diarrhœal fatality is closer with the number of flies in circulation than with any other fact. The next closest connexion is with the readings of the 4-ft. thermometer, with which, however, diarrhœal fatality can have no direct relation. Flies and the readings of the 4-ft. thermometer are both functions of air and surface temperatures and of rainfall.

Certain facts in the life-history of the fly throw light on discrepancies arising in the decline of flies and cases. The close correspondence between flies and cases of fatal diarrhoea receives a general support from the diarrhoea history of sanitary subdivisions of the Manchester district. The few facts available for the study of the correspondence of flies and fatal cases in different subdivisions, in the course of the same year, also lend support to this view. No other explanation even approximately fits the case.

ENTERIC FEVER AND FLIES.

At this point it will be convenient to give the main table, on which the facts are based. Table A gives for each year since 1891 the number of deaths from diarrhoea in each of the fifty-two weeks of the year, missing out the odd week in leap year. It also gives the cases of enteric fever reported to the Public Health Office, arranged not according to the number of cases notified in each week, but according to the number of cases commencing in that week. The dates of commencement are founded on the reports of the District Sanitary Inspectors, and are therefore to some extent imperfect, except when special skill is employed. The results show, however, that these dates are not far out, and it is to be remembered that the reports are annotated and revised by skilled examiners, while the Inspectors also have been instructed on the principles according to which they are to fix the dates of commencement. These data both for diarrhoea and enteric fever are added up in corresponding weeks of successive years to give a composite series in two periods, 1891 to 1897, and 1898 to 1908. This is done because the notifications, so far as enteric fever is concerned, are checked in the later period by the serum test, and are much more reliable in consequence. But this division has the incidental advantage that it permits comparison of two different periods. The figures for individual years are illustrated by charts. In these the number of deaths from diarrhoea has been divided by five for convenience of representation (*see* pp. 192 to 199).

TABLE A.—DIARRHŒA DEATHS (IN WEEKS).

Year	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
1891	4	2	3	4	4	2	4	3	3	3	8	1	—	2	3	12	6	4	5	5	1	4	2	5	4	10
1892	2	2	2	2	—	1	3	3	3	5	4	2	2	3	7	1	1	2	3	2	2	1	2	2	2	9
1893	—	2	2	4	9	1	1	4	7	4	3	7	4	2	6	4	2	3	4	7	5	4	5	11	19	58
1894	5	3	5	5	3	1	3	5	1	3	8	—	5	7	8	3	5	3	3	1	6	4	3	7	1	5
1895	2	7	4	4	3	5	3	4	5	4	4	1	5	2	5	1	5	1	7	6	4	1	3	9	5	8
1896	4	3	1	4	6	5	1	1	5	—	2	3	3	3	8	6	3	—	3	4	5	5	7	8	10	16
Total	17	20	21	23	19	15	15	20	24	19	29	14	19	19	37	27	22	13	25	25	23	19	22	42	41	106

Year	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52
1891	7	9	5	13	8	15	19	18	17	33	29	21	28	17	14	14	8	7	4	3	3	4	2	2	1	4
1892	4	7	3	3	15	17	24	46	49	33	35	17	12	14	17	4	4	3	6	3	4	8	3	2	2	4
1893	72	83	92	60	38	31	39	44	51	39	37	30	25	16	22	8	3	9	6	7	3	1	2	—	5	3
1894	9	12	15	16	20	28	31	18	16	14	9	6	7	5	7	9	9	8	3	2	2	3	3	1	7	1
1895	5	9	21	24	29	31	46	62	60	68	74	60	59	55	48	30	28	17	12	5	8	5	3	4	3	3
1896	18	11	42	58	61	47	46	19	27	21	11	8	10	11	2	7	6	7	2	6	5	1	3	8	3	2
Total	115	131	178	174	171	169	205	207	220	208	195	142	136	118	110	72	58	51	33	26	25	22	16	17	21	17

DEATHS FROM DIARRHŒA.

Year	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
1897	1	7	6	2	7	2	2	3	5	5	1	3	5	7	1	3	1	4	3	4	5	3	5	4	4	5	8	7
1898	5	4	1	3	3	6	6	2	5	3	7	3	6	2	2	3	3	4	2	2	4	6	2	7	6	6	3	9
1899	4	3	2	5	3	2	6	8	2	8	2	4	4	1	1	2	4	1	3	4	5	4	4	9	4	8	5	9
1900	3	3	3	4	3	4	—	7	3	3	3	1	3	4	8	3	4	6	5	7	5	4	1	7	6	7	6	7
1901	2	4	4	6	1	6	1	5	7	3	2	4	—	5	4	3	3	3	3	1	2	3	3	1	3	—	3	12
1902	7	3	2	2	3	2	—	3	1	2	3	1	3	3	3	—	—	2	7	1	4	5	1	4	5	3	1	1
1903	3	4	2	5	8	4	5	2	3	5	4	2	1	1	1	—	4	3	3	4	4	6	2	4	5	9	6	13
1904	5	1	2	4	1	1	2	2	1	2	2	1	3	1	5	4	3	6	1	1	1	—	4	5	4	4	7	—
1905	2	5	3	3	—	—	—	1	1	3	2	—	3	1	1	3	—	6	1	1	1	1	—	4	5	4	4	7
1906	1	2	2	2	3	3	2	5	3	2	3	—	4	3	2	4	2	5	3	3	1	2	5	3	1	3	4	6
Totals	33	36	27	36	32	30	24	32	38	32	31	22	31	33	28	25	22	44	27	31	33	32	30	46	40	48	48	71
	132												117				124				141				207			
	118												117				124				141				207			

Year	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52
1897	15	33	71	150	167	116	73	65	46	28	18	5	8	10	4	4	4	6	5	4	2	2	1	6
1898	5	20	36	54	46	46	87	111	108	111	121	110	50	40	26	14	14	7	6	6	5	4	3	4
1899	22	42	75	103	107	136	128	129	81	79	32	16	9	4	11	5	2	6	4	4	3	5	4	1
1900	11	13	37	63	50	50	83	78	64	36	58	41	26	19	20	15	4	6	5	5	2	3	4	3
1901	36	79	122	119	107	110	86	68	61	35	26	21	12	7	8	5	4	2	2	5	3	—	2	2
1902	3	4	3	3	2	2	10	14	19	40	15	20	17	14	8	6	14	5	6	4	3	4	1	8
1903	14	14	23	29	39	42	31	21	24	20	26	20	22	11	7	9	6	6	2	4	10	4	1	4
1904	14	48	77	91	107	85	68	43	36	28	24	23	11	3	3	4	1	—	3	3	2	4	3	3
1905	22	62	81	89	67	81	61	61	42	20	15	10	9	5	4	6	3	3	5	5	4	1	5	3
1906	6	12	17	34	80	102	104	135	136	86	58	35	28	11	13	16	9	5	2	2	4	3	1	3
Totals	148	327	542	741	772	770	731	725	617	483	393	301	192	124	104	84	61	46	37	40	39	33	21	39
	1758												504				184				132			
	2998												1794				184				132			

TABLE A (continued).—ENTERIC FEVER COMMENCING IN EACH WEEK OF THE YEAR, FOR THE YEARS 1891 TO 1896.

Year	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
1891	11	10	12	18	9	18	15	8	15	10	9	14	16	15	13	15	16	12	7	8	6	8	8	6	4	1
1892	13	17	15	13	14	12	8	13	14	13	8	10	14	9	3	6	6	17	9	14	6	11	2	6	5	12
1893	12	10	10	13	8	5	6	9	6	9	6	9	5	10	5	8	1	5	2	4	5	3	9	10	11	7
1894	12	6	11	15	7	13	5	11	9	8	3	9	9	12	6	5	10	9	2	4	3	5	2	4	7	6
1895	8	7	12	7	15	11	9	9	10	4	5	9	4	2	4	6	9	8	6	7	10	13	8	6	6	5
1896	11	8	9	17	10	9	8	9	8	12	9	6	2	8	13	17	10	18	7	10	10	4	9	9	11	4
Total	67	58	69	83	63	68	49	59	62	56	40	57	50	56	44	57	52	69	33	47	40	44	38	41	44	35

Year	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52
1891	4	7	8	5	8	12	14	25	22	20	18	19	18	32	23	28	17	22	25	26	35	15	20	18	15	13
1892	4	6	10	12	9	8	9	11	20	13	19	13	22	17	7	11	15	27	19	14	16	23	9	11	10	5
1893	13	10	10	10	15	14	14	20	19	28	25	27	15	19	15	30	27	18	20	20	14	14	9	9	9	5
1894	7	6	4	6	11	13	17	14	17	9	6	11	14	9	12	10	10	11	12	11	15	10	10	5	6	11
1895	3	6	3	4	8	6	8	9	6	8	12	8	19	17	16	12	17	25	21	19	20	9	10	15	6	6
1896	5	10	7	5	7	8	8	11	6	14	12	4	11	11	18	18	17	13	10	6	9	10	15	6	8	9
Total	36	45	42	42	58	61	70	90	90	92	92	82	99	105	92	109	103	116	107	96	109	81	73	64	54	49

CASES OF ENTERIC FEVER COMMENCING IN EACH WEEK OF THE YEAR, FOR THE YEARS 1897 TO 1906.

Year	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
1897	10	10	7	5	10	5	8	7	11	6	8	6	4	5	10	3	2	3	5	3	9	1	4	3	3	6	4	3
1898	13	12	19	14	12	12	9	15	12	9	8	2	6	9	10	14	10	11	4	2	1	3	6	4	1	4	1	7
1899	12	14	16	12	5	9	6	5	3	5	6	3	5	7	6	7	10	3	1	6	4	6	3	4	4	4	2	—
1900	11	8	7	10	2	7	5	7	4	5	3	10	6	12	11	7	8	8	4	3	4	2	7	5	6	5	2	—
1901	4	3	3	3	3	5	4	—	4	2	2	4	6	2	8	2	6	2	4	1	2	1	5	5	3	2	2	—
1902	3	3	3	6	5	7	6	7	5	6	5	7	7	8	9	11	7	2	4	4	4	3	8	4	10	8	1	2
1903	8	3	4	5	8	4	11	7	5	6	9	6	14	10	4	4	3	1	6	7	14	7	3	12	5	6	5	3
1904	3	5	9	11	1	3	5	3	6	8	4	4	7	7	3	10	10	6	5	6	2	4	3	3	7	2	3	6
1905	9	11	7	12	12	3	5	5	9	10	12	9	7	7	11	5	6	8	6	4	4	1	1	4	3	2	1	1
1906	7	14	15	14	7	4	2	6	2	3	4	2	1	3	1	3	8	3	—	3	3	3	2	4	5	3	9	4
Totals	80	83	90	92	65	59	61	62	58	56	61	53	63	70	73	66	70	47	39	39	46	31	42	48	47	40	33	29
	345				247				228				272				195				167				149			

Year	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52
1897	6	5	3	8	7	17	20	12	13	19	20	16	17	11	12	13	18	12	20	21	20	17	23	12
1898	4	4	6	7	8	7	23	15	13	18	21	28	39	31	30	21	10	20	17	20	20	18	14	18
1899	2	5	5	5	15	11	12	10	12	10	15	12	6	9	7	10	8	9	10	9	10	4	7	9
1900	3	6	7	9	9	8	13	10	12	9	17	14	19	11	10	10	5	12	7	8	9	3	1	3
1901	3	7	13	15	13	12	13	22	14	20	14	17	27	11	15	9	9	9	6	7	3	6	2	3
1902	4	9	10	6	5	8	10	7	5	17	20	11	9	12	12	10	3	10	5	14	7	14	5	10
1903	6	6	6	6	6	6	5	4	6	11	14	12	13	9	5	16	15	9	8	9	11	9	7	5
1904	6	6	9	8	7	9	6	10	13	7	18	7	7	4	7	9	5	7	6	6	5	7	12	1
1905	—	7	2	4	9	8	6	6	7	11	7	13	6	12	9	10	4	10	4	6	6	8	6	7
1906	5	3	7	2	6	4	4	13	20	19	14	13	22	16	23	16	7	14	9	8	8	2	6	8
Totals	39	58	68	70	85	90	114	109	115	141	160	143	165	126	130	124	84	112	92	108	99	88	83	76
	235				398				559				645				396				346			

Before entering on a consideration of these data it is necessary to sum up the results of previous work in connexion with enteric fever. This disease has been treated of in the Manchester Annual Reports from 1896 onwards, but the first extensive analysis was made in the years 1904 and 1905, and these, with the subsequent reports, will be found to contain data which will probably be useful to those desirous of tracing the course of an outbreak in an urban community. In 1883 an account of an outbreak in Newton Heath, Manchester, will be found in the *Edinburgh Medical Journal* for May and July,¹ in which special stress is laid on the extent of infection between individuals in contact, and on the considerable proportion of very slight attacks which might easily have been overlooked. In a paper in the *Manchester Medical Chronicle* of 1887, entitled "Remarks on Fever,"² in explanation of the comparative severity of incidence observed on new-comers to a district in which typhoid fever is present, it is said: "It is true that the numbers attacked are not a complete proof that the new-comers suffer more severely than the residents, since we know that many cases of typhoid fever, especially in children, escape altogether." The observations made in 1904, 1905, and 1906 confirm these remarks. As in previous and subsequent reports, the histories of what one may call "contact" infection are given very fully. In 1904 between one-fourth and one-fifth of the cases are traced to such infection. In 1905 over one-third are linked together, and in this year the cases thus connected are, by the skill of the inquirers, grouped in nests. In 1906 the cases are not published in detail, but it is stated that 36·8 per cent. are traced to contact with previous cases. In 1905 and 1906 a free interpretation of the word "traced" must be allowed, although the facts are fully displayed in the annual report for the former year. Thus, with sufficient energy and skill, the sources of enteric fever may be much more fully ascertained than they usually are. That we have been able to do so is due chiefly to the acumen of a particular Inspector, Mr. Hewitt. Needless to say, the alleged cases brought to light in the course of the inquiries in 1905 are mostly confirmed by the serum test.

In the annual report for 1904, from an analysis of the periods in the disease at which cases were notified by practitioners, I was able to assert that a large number of cases must escape attention altogether. These inquiries fully prove this directly, as is seen from the following figures for 1905:—

	Aged under 5	Aged 5—9	Aged 10—14	Aged 15 +	Total
Enteric fever, all cases ...	27	29	38	217	311
Cases discovered by Mr. Hewitt ...	11	5	6	13	35
Cases discovered by other inspectors	8	4	3	16	25

¹ *Edin. Med. Journ.*, 1883-4, xxix. p. 121.

² *Med. Chron.*, Manchester, 1887, vii, pp. 1, 199.

Thus, about one-sixth of all cases known to us had been overlooked. These overlooked attacks are, it will be seen, specially liable to occur in children, and, of these, in young children. A number of them are infants apparently suffering from diarrhoea. In the 1904 report it is recorded that of twenty-six overlooked cases causing infection, six were believed to be suffering from diarrhoea. It is evident, then, that there is a considerable amount of cloaking of enteric fever by diarrhoea in the diarrhoea season. Further, these overlooked cases have, as we might expect, a special influence in causing infection. In 1904 there were twenty-six overlooked cases giving rise to subsequent attacks, of which nine caused thirty subsequent. In 1905 there were thirty-six ascertained overlooked attacks, giving rise to subsequent attacks. The whole subject is reviewed in the Manchester Annual Report for 1907, to which I would refer.

In the above statement nothing has been said about the manner in which disease is conveyed from one person to another. This is usually assumed to be through contamination of food, although it is often difficult to see how this could have occurred, unless there are still cases which we have overlooked—cases, in fact, so slight that they elude clinical inquiry altogether. There can be practically no doubt that there are many such cases. Amongst other facts it may be mentioned that at various times we have subjected to the serum test all members of families, and in some instances alike those who have been markedly ill, those who have had slight illnesses, and occasionally those who had apparently not been ill at all gave well-marked Widal reactions. Even in the absence of such cases, however, it does not necessarily follow that infection should be conveyed by handling food. In the season of house flies these may act as carriers, or some other living agents of transmission may be at work. If these are capable of carrying infection from one house to another, they are a priori capable of transmitting it within the house. We are thus led to inquire as to the proportion of "contact" cases occurring in different seasons. This point is investigated for the years 1905 and 1906, in which we find the following proportions:—

1905.										
	First quarter		Second quarter		Third quarter		Fourth quarter		Total	
Cases traced with precision to direct contact	38	...	13	...	10	...	11	...	72	
Cases associated with the previous consumption of mussels	26	...	20	...	20	...	31	...	97	
Untraced	32	...	22	...	37	...	55	...	146	
Total	96	...	55	...	67	..	97	...	315	

¹ Of the 315, 4 are either not enteric fever or are doubtful.

1906.															
			First quarter			Second quarter			Third quarter			Fourth quarter			Total
Cases traced to direct contact	38	7	17	64	126
In connexion with shellfish	37	15	17	37	106
Untraced	15	7	35	55	112
			—			—			—			—			—
Total	90	29	69	156	344

In both years the proportion of cases traced to *direct contact*, which for the most part means occurring in the same household or from visiting an infected house, is low in the third quarter, a circumstance which may be variously interpreted.

The next most important factor in the propagation of enteric fever, at any one time, is the eating of shellfish, and by shellfish in Manchester is meant mussels. Enormous quantities are consumed in the open season, from September 1 to April 30, the largest quantity being used at the commencement of the season in September and October, and up to the close of the Christmas holiday. A fair quantity is also used in the close season, from April 30 to September 1, at which time they are brought from Scotland and Ireland. It is, however, certain layings off our own coast which are more especially associated with the occurrence of enteric fever. They are brought from Liverpool to the wholesale dealers, or large retailers, in bags. By these they are consigned to retail shops, or purchased by hawkers, who, no doubt, buy the poorer qualities. The large quantities sold by hawkers we have no means of tracing. When the mussels, suspected in connexion with a case, have been bought from a retail shop, we can often ascertain the date on which they were purchased, and the wholesale man. From him we get the laying. In this way we have been able by accumulation of facts from year to year to build up a case in respect of the layings from which infected mussels come. Occasionally more than one person contracts enteric fever from eating of the same mussels at the same time. In this and other ways we have reached the conviction that mussels are responsible for a good deal of enteric fever. It does not follow, of course, that because a man who has eaten mussels begins with enteric fever, a week or a fortnight or three weeks afterwards, the mussels have been the source of his attack. It is probable, however, that a considerable number of such connexions are linked as cause and effect. Of the cases arising in connexion with shellfish in 1904, we find that notwithstanding the large number traced to direct contact, scarcely any of those found to be associated with mussels belonged to the groups which had been exposed to direct contact. This is also true of 1905.

Inasmuch as mussels are mostly eaten by adults and adolescents, it is to be expected that the distribution of cases associated with their consumption would be different from the distribution of those traced to contact. The following table from the annual report for 1904 shows the mode of distribution according to sex, age, and employment :—

Occupations		All cases		Traced definitely or probably to contact with a previous case		Having consumed shellfish raw or cooked before attack
Workers in cloth, clothing, &c.	...	43	...	5	...	6
Nurse, laundresses, charwomen	...	7	...	1	...	0
Provision dealers, &c.	...	18	...	0	...	9
Ironworkers and their labourers	...	23	...	1	...	9
Other labourers	...	21	...	2	...	5
Other occupied males	...	36	...	3	...	7
Housewives	...	45	...	14	...	7
Children under 14	...	94	...	36	...	3

The sex and age of cases associated with the consumption of shellfish may be seen also from the following figures for the years 1904 and 1905 :—

1905.													
	Aged 0—14		Aged 15—24		Aged 25—34		Aged 35—44		Aged 45—64		Aged 65 +		Total
Males	...	5	...	24	...	28	...	10	...	1	...	0	68
Females	...	6	...	7	...	13	...	3	...	2	...	0	31

1904.													
Males	...	4	...	8	...	16	...	7	...	1	...	0	36
Females	...	1	...	4	...	2	...	2	...	2	...	0	11

In 1906 there were 106 cases associated with consumption of shellfish; males and females under 15 numbered 10, males aged 15 and upwards 73, females aged 15 and upwards 23. In 1907 there were 67 cases, in 1908 88 cases, 71 of whom were in males and 17 in females. It thus appears that the occurrence of cases due to shellfish is a factor in determining the excess of incidence on males. An analysis of the seasonal incidence of enteric fever in the Manchester Annual Report for 1907 leads to the same conclusion. Yet the excess still remains when the shellfish cases are taken away. The consumption of infected shellfish is not, therefore, the sole cause of the excess of incidence on males.

The above figures, of which those for 1905 and 1906 represent the most complete investigations, give an estimate of the cases which may be due to shellfish. If we assume that the drop in the nineteenth week which lasts during the summer represents the withdrawal of the shellfish influence, we may form some estimate of its amount. Now, the drop on the average of ten years, 1897 to 1906, about the nineteenth week, will be hereafter seen to be 3·3 cases per week. If we may assume the excess

to last seventeen weeks, we get 56 cases due to mussels after the new year. The number prior to that date will be somewhere about $\frac{3}{2} \times 56$, or 84. The total average number due to shellfish, among notified cases, would thus be 140. The average number proved to be connected directly with consumption of shellfish in 1904-1908 is

$$\frac{47 + 99 + 106 + 67 + 88}{5} = \frac{407}{5} = 81$$

The difference of 60 could be considered as expressing the cases indirectly due to shellfish.

It is probable that a considerable number of persons eat mussels when they have had too much liquor. These are purchased from hawkers, eaten and forgotten; contaminated mussels, therefore, probably do more harm directly than we can trace. The contaminated batches come at irregular intervals, and the cases arising from them will be liable to rise and fall in an irregular manner. The quantity consumed is, however, much greater in the period before Christmas than from that date to the end of the season. A study of the circumstances will show that the maximum infection from this cause will be prolonged at least into October and November, depending as it does on infection from the riverside towns above the layings. Not a few infections have been traced to a primary mussel-infected case, and infected shellfish thus exert in Manchester a marked influence in sustaining the continuance of enteric fever.

In considering the modes of propagation of enteric fever there are other points of view which we must think of. Attacks in males preponderate considerably over attacks in females. I have given the following figures for 1904. From the census returns we get the numbers of persons living in Manchester at the census of 1901 :—

Males aged 14 and upwards (these include unoccupied males)	...	181,172
Occupied females, aged 14 and upwards	...	86,978
Unoccupied females, aged 14 and upwards	...	110,878
Children under 14 years of age	...	164,844

ENTERIC CASES OCCURRING IN MANCHESTER IN 1904.

Workers (male) aged over 14 (these include cases in unoccupied males)	141
Workers (female) aged over 14	47
Women employed in housework, &c.	54
Children up to age of 14	77

The greatest incidence is on males at ages above 14, then on female workers, then on unoccupied females, least on children. The usual explanation is that many cases of enteric are ambulant, and work

increases the opportunities of infection ; then, of course, workers have money to spend, and can eat raw mussels and other unwholesome dainties as they choose. The inquiry into the influence of specific occupations reveals nothing of importance. As we have seen, there is reason to believe that the incidence on younger ages is quite understated by our public records, if uncorrected. As regards social station, it was shown in the annual report for 1904 that enteric fever is a disease of the artizan and poorer classes. Malnutrition appears decidedly to predispose to it.

The water supply as it arrives in Manchester has been shown by Professor Delépine, and in my annual report for 1904, not to be responsible for the spread of enteric fever. The milk supply must also be exonerated so far as our records of individual cases go. Watercress of the cheaper sorts may produce some effect in March and April, at which season there is a slight swell in the enteric wave. Fruit may be contaminated by flies, as may other articles of consumption, and convey the disease in the fly season.

Further light may be got from tables or spot charts showing the occurrence of cases for each month or week in each of the eighteen sanitary districts of the city. These spot charts will be found in the annual reports for 1904, 1905, and 1906 ; the corresponding facts will be found for 1900 to 1903 in the annual report for 1904, and may for these years be exhibited as spot charts.

From a study of these facts and charts in the annual report for 1904 the following conclusions were drawn :—

- (1) Strictly local rises are apt to occur at all periods of the year.
- (2) They generally extend over two or three months, but may be limited to one, in which case infection in one family may be the cause.
- (3) Such local rises occur in the first six months of the year 1900 in five districts, in 1901 in one district, in 1902 in seven districts, in 1903 in six districts, and in 1904 in four districts.

The above statement made in 1904 contains much of what there is to be said with reference to the autumnal rise of enteric fever, which is nevertheless greater, as a rule, than these remarks would indicate. In subsequent reports greater inclination exists towards recognition of the action of the house fly as the cause of the autumnal rise than is there shown. The facts for individual years vary greatly. In some years the increase in cases accompanying and following the diarrhoeal wave is very striking. The autumnal rise may be divided into two parts—viz., that which precedes and that which follows the thirty-eighth week. In

Manchester it is not possible to separate in this and subsequent weeks the influence of mussels from other factors, and the curve now represents a blend of influences. It is, however, occasionally possible to see the part of the curve subsequent to the primary enteric rise separated off from that which is influenced by mussels. And it then behaves much as if there were no mussels. Now the primary part of the autumnal rise exhibits in most years a very striking phenomenon. In some one week the cases of enteric suddenly increase, and this increase is maintained with subsequent further but very irregular ascents and descents. The tendency of the primary rise is to have a flat top for some weeks, a tendency which comes out very strongly on adding the facts for different years. This tendency to flattening is characteristic of slight effects, the period of occurrence of which cannot be definitely fixed. It is very manifest in the diarrhœa curves in 1909, and in the diarrhœa cases commencing day by day in years of severe incidence. It is dependent on want of precision in dates of onset, in latent periods, and so forth. Nevertheless, though not great, the increase usually occurs in a decisive and striking manner. This first part of the enteric autumnal rise is that which we have to explain, though we cannot overlook the subsequent part. In the valuable contribution made by Dr. Sandilands to the question of the infectiveness of diarrhœa, he adduced evidence to show that in towns in which middens had been converted into water-closets a much greater and a more characteristic fall had occurred in the incidence of enteric fever than was manifest in other towns which retained their midden-privies. On the face of it, this would seem to be due to conversion of these closets into water-closets. Certainly, however, a great change may occur, and has occurred in Manchester without such conversion. In 1891 the death-rate from enteric fever was 0·37 per 1,000 persons living. In 1899 it underwent a sudden fall to 0·12 per 1,000, and fell further with oscillations, till in 1907 it was 0·06 per 1,000. In 1908 it was 0·12 per 1,000. Now, the drop in 1899 was not due to conversions, which are now proceeding rapidly, but were not in progress at that period. It is true, special pails began to be supplied in 1894 to houses in which enteric fever occurred, and a marked fall was witnessed in that year. Further, processes of disinfection of middens and pail sites became more active after that year. Nevertheless, this would not meet the difficulty of overlooked cases. It is also to be remembered that the development of our towns for many years has meant a relative increase of water-closets. At the same time there can be no doubt that conservancy systems, and

especially privy-middens, have a decided influence on the continuance of enteric fever. From figures which I prepared for the years 1891 to 1898 it appeared that the proportion of cases occurring in connexion with middens to those occurring in connexion with pails increases in the aggregate of years, though not in every year, in the months of August, September, and October, the season of flies.

At this point we may consider why flies should be thought of in connexion with midden-privies. They are, of course, well known to visit these places and to breed there. But, in addition, as we have seen, enteric fever is more severe on males than on females, and on female workers than on housewives, and (according to the figures) than on children. But the housewife or one of the children empties the ashes, cleans the closet, and performs all those duties which bring members of the household into direct relation with the privy. It seems likely, therefore, that the influence exerted *by* the midden is not exerted *at* the midden, but consists in something which is transferred to the house and affects all the members of the family alike. Such an influence would be the transmission to food of infection from the midden by the house fly or by some other living pest. This follows, unless we regard the influence of home life in determining sex-incidence as being an altogether subordinate one, a view which the ascertained facts would seem to contradict. We must, however, take into account also the greater tendency to treat females and children at home, while the breadwinner, when disabled, is to a much greater extent notified and removed to hospital. This tendency is very manifest in the case of phthisis.

STUDY OF THE INCIDENCE OF ENTERIC FEVER IN ST. GEORGE'S SANITARY DISTRICT IN 1904.

A map is given in the above year of the occurrence of cases in this district and in Harpurhey, showing four distinct nests of enteric fever in St. George's and one in Harpurhey. St. George's is served by pail-closets, Harpurhey partly by middens. Many of the cases in St. George's are linked together by histories of contact. The occurrence of these nests in St. George's in place and time excludes house flies and shellfish as main factors. The nests are probably determined in some way by local infections of food, and are probably of the nature of contact infection. To some extent also this is true for Harpurhey. In 1906, however, a fresh and more intense outburst occurred in Harpurhey and Moston further along the Rochdale road, which was in the fly season, and in which midden-privies probably played an important

part. This study serves to emphasize the influence of contact infections, not always clear in their character, and shows that our investigations still leave obscurities to be cleared up. These local nests of enteric fever are to be distinguished from the infective nests in the annual report for 1905, in which the cases were often some distance apart.

We may sum up these observations as follows: There are two proved influences operating to produce the enteric curve in Manchester—so-called direct contact and mussels. The influence of both is powerful, that of the second problematical only in amount. It is, however, limited in time, and hence, on the addition of the facts for different years, this influence should be clearly manifested. Owing to the commencement of the close season for mussels on April 30, there should be a fall observed in the added figures on the nineteenth week, a fall maintained during the rest of the year up to about the thirty-eighth week, in which and in the following weeks we may suppose the effect of the mussel season to be first and most markedly manifested. If flies cause the transmission of enteric fever, we may expect that the fall occurring at the close of the mussel season will remain until the fly season has begun. Owing, however, to the comparatively small amount of enteric infection to which flies have access we may expect that the enteric rise will be later than the diarrhœal. Further, as enteric fever has so greatly declined, we should anticipate that a much larger number of flies would now be required to affect the enteric curve than was the case in 1891. Hence the enteric rise will be shifted back, as we go backward in years, relative to the diarrhœa curve. It will also have a much increased tendency to disappear in years of low diarrhœal fatality, and therefore of low numbers of flies. If this should be found to happen irrespective of conditions of temperature or other states of weather, we shall have strong reason for believing that flies do cause the primary rise of enteric fever in autumn. Then again, as we have seen, there is good reason to believe that many cases of enteric fever are regarded as diarrhœa when the latter disease becomes prevalent. This is more particularly true of years of high diarrhœal death-rates, although, owing to the comparatively low amount of enteric fever present at all times, the amount of this error will not increase in proportion to the severity of diarrhœa.

We may therefore look for two increases in enteric fever following the fly season, a primary increase following close on the increase in diarrhœal fatality, and a subsequent increase due to contact infection from overlooked attacks of enteric caused by flies and recorded as diarrhœa—an increase not necessarily small, in the light of the histories

of "direct infection" from overlooked cases which have been accumulated. There is a third source of increase at the later period. The turnover of diarrhoeal infection, if one may use the expression, is rapid as the curve of flies ascends. That of enteric fever is much slower. There will, however, in general be enough flies left, when the primary rise has occurred and when the overlooked cases come into play, to produce a secondary direct fly increase. These two secondary fly increases will in general come into collision with the mussel increase and we must disentangle them as best we can.

If now we refer to the figures showing the number of cases of enteric fever added week by week for the years 1897 to 1906, we find that a drop occurs in the nineteenth week, and this fall is sustained up to the thirtieth week, when a slight rise is manifest. The numbers reported week by week then advance irregularly, marked increase occurring in the thirty-third week. A second marked increase occurs in the thirty-fifth week, and thereafter the curve remains level. A third jump upwards, and the greatest, occurs in the thirty-eighth week, and is sustained for three weeks. A marked fall occurs in the forty-second week, the curve remaining at about the same level to the forty-fifth week, after which it declines irregularly up to March. A small rise occurs from the thirteenth to the seventeenth week, after which the numbers fall to their summer level. Now it appears certain that the summer fall in Manchester is conditioned by the withdrawal of contaminated mussels from the market, mainly, though not entirely. The abrupt fall in the beginning of May must certainly be ascribed to this cause. Similarly the rise in the thirty-eighth, thirty-ninth, and fortieth and forty-first weeks is largely due to the commencement of the mussel season. Is the rise from the thirtieth to the thirty-seventh week to be ascribed to flies? Referring to the figures for diarrhoea, we see that in the added series flies must have been at a maximum from the thirtieth to the thirty-fifth week. We may surmise, then, that much of this part of the curve is due to direct infection by flies. By the end of this period, however, the number of cases of enteric fever notified weekly has increased threefold, while the number of flies has by no means diminished proportionately. Not only so, but there are also numerous overlooked centres of infection. It may be, then, that the influence of flies alone would produce a rise in this part of the curve, apart from mussels altogether. This question we are in a position to put to the proof. If this be so, taking into account the tendency of enteric fever to spread by direct contact in nests, having regard also to the probability of

overlooked cases increasing *pari passu* with cases due to flies, and taking into account the continuance of flies to a late part of the season, we might suppose that we had explained the enteric curve in Manchester.

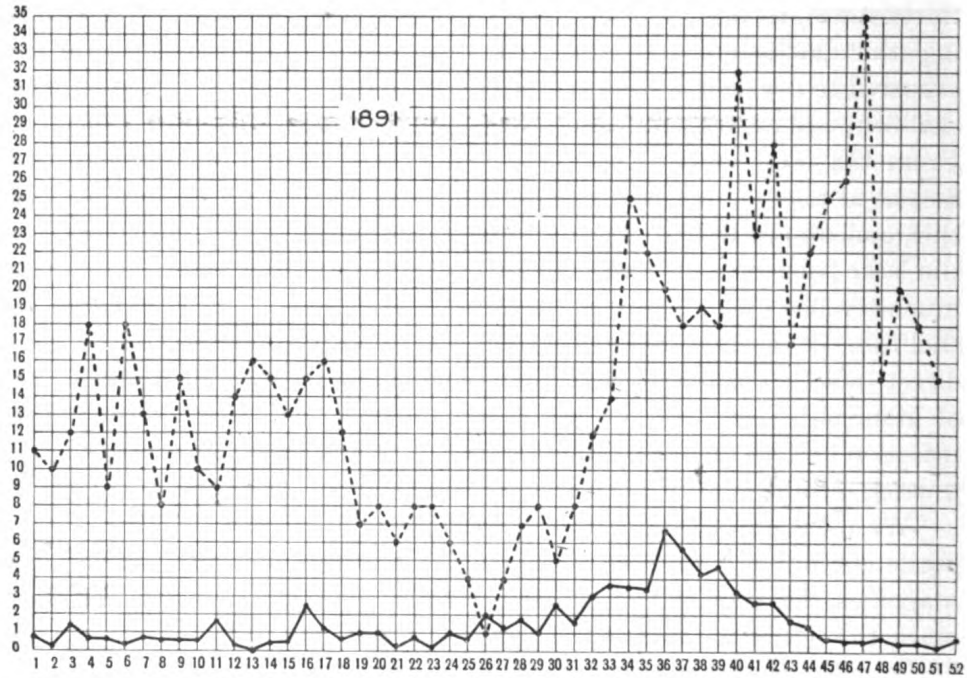
We are in a position to eliminate any influence due to shellfish in the years 1904, 1905, and 1907. We get for these years the following numbers in weeks of cases of enteric commencing, of cases associated with consumption of shellfish, of the numbers found by subtracting these, and for the last half of the year of deaths from diarrhœa:—

Weeks	Number of cases of enteric commencing	Number associated with shellfish	Difference	Diarrhœa deaths
1	33	14	19	—
2	25	12	13	—
3	26	4	22	—
4	33	6	27	—
5	27	4	23	—
6	17	3	14	—
7	27	5	22	—
8	17	7	10	—
9	27	9	18	—
10	32	2	30	—
11	27	10	17	—
12	23	4	19	—
13	34	5	29	—
14	24	3	21	—
15	18	4	14	—
16	19	6	13	—
17	19	7	12	—
18	15	4	11	—
19	17	5	12	—
20	17	2	15	—
21	20	3	17	—
22	12	4	8	—
23	7	2	5	—
24	19	4	15	—
25	15	1	14	—
26	10	—	10	—
27	10	1	9	24
28	10	1	9	26
29	13	1	12	59
30	20	1	19	144
31	18	0	18	203
32	20	2	18	259
33	20	2	18	296
34	33	3	30	281
35	23	2	21	217
36	30	1	29	154
37	34	8	26	128
38	36	6	30	89
39	47	15	32	86
40	45	9	36	73
41	36	6	30	54
42	33	9	24	37
43	28	6	22	32
44	41	10	31	36
45	37	4	33	16
46	35	10	25	14
47	37	8	29	8
48	53	9	44	13
49	53	9	44	19
50	43	12	31	16
51	42	12	30	9
52	24	8	16	12

We thus see that though the rise in the thirty-seventh, thirty-eighth, thirty-ninth, fortieth, and forty-first weeks is much reduced by subtracting the shellfish cases, it is not removed. We must therefore be prepared to consider it an integral part of the enteric curve, apart from shellfish, and as probably associated with flies in the manner indicated. From the forty-fourth week onwards we must regard the influence of flies as at an end, and we expect a decline of incidence. We do not get this, and we are thus inclined to doubt the adequacy of flies to explain the later parts of the curve. In the same way we find that the curve is much levelled about the nineteenth week by removing the influence of shellfish. But some difference remains. The impression remains that direct infection of the kind exhibited in the annual reports on the health of Manchester for 1904 and 1905, taken along with the conveyance of infection by flies and the reinforcement of the disease by contaminated mussels, almost explains the whole phenomena of enteric fever.

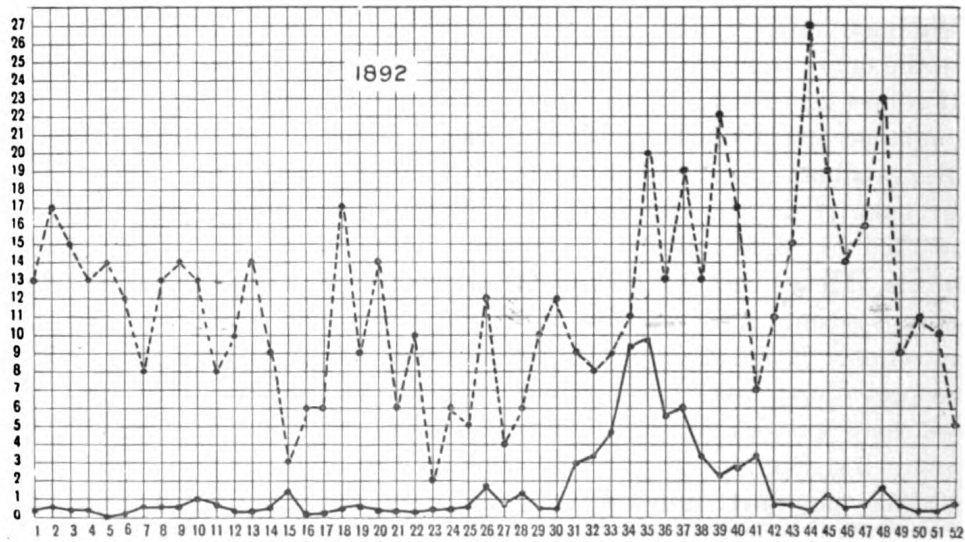
We must now consider the individual curves of enteric. The composite curve which we have just considered brings into prominence and magnifies the influence of mussels on the course of enteric fever, but blurs the action of flies which occurs at different periods in different years. We thus get some measure of the amount by which mussels affect the course of the disease. The number of cases in the eighteenth to the twenty-first week of the ten years' series averages 43, in the four weeks preceding 70. There is thus a drop of 33, averaging 33 per week on the ten years. A similar rise occurs in the thirty-eighth, thirty-ninth, fortieth, and forty-first weeks, which, however, is now due in part to flies. We may, however, obtain an idea of the magnitude of this contribution to the curve from the figures already given. It is clearly considerable. It comes in rushes. Like the result of fly infection, each access is no doubt accompanied by overlooked cases. Contact or direct infection also produces nests limited in time. Both influences tend to produce irregularity in the curve. The influence of fly infection will not be marked by such ups and downs, and will proceed more smoothly, as a rule. Hence that part of the curve which is most marked by irregular, abrupt ascents and descents is most likely to be due to mussels or direct infection. If the ascents occupy three weeks or upwards we may usually suspect direct infection or mussels; if one week only, and very steep, we may suspect mussels. This is, however, a very imperfect first approximation.

We may next examine the figures and charts for separate years—1891 to 1908. The following observations may be made on the series: Enteric fever cases commencing in weeks are shown as a black dot on the

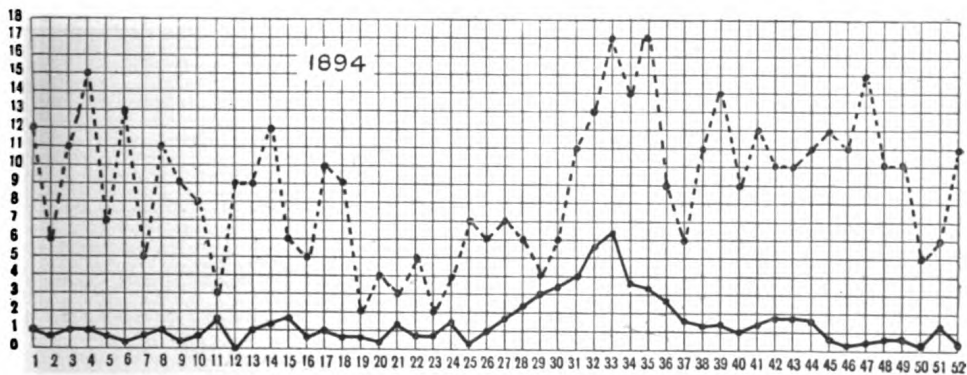
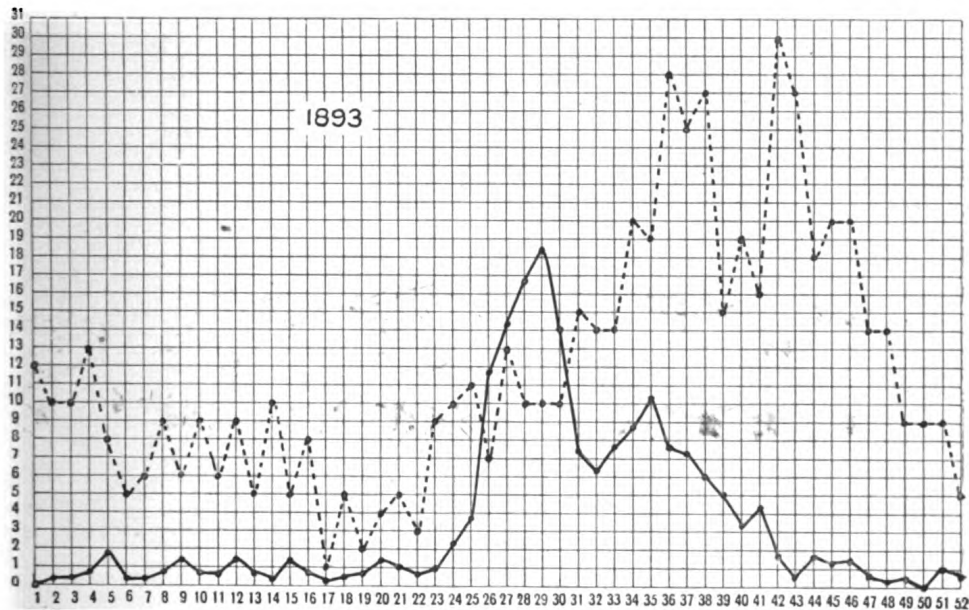


In this and the following seventeen charts

— indicates diarrhœa deaths, divided by five
 ,, enteric cases commencing

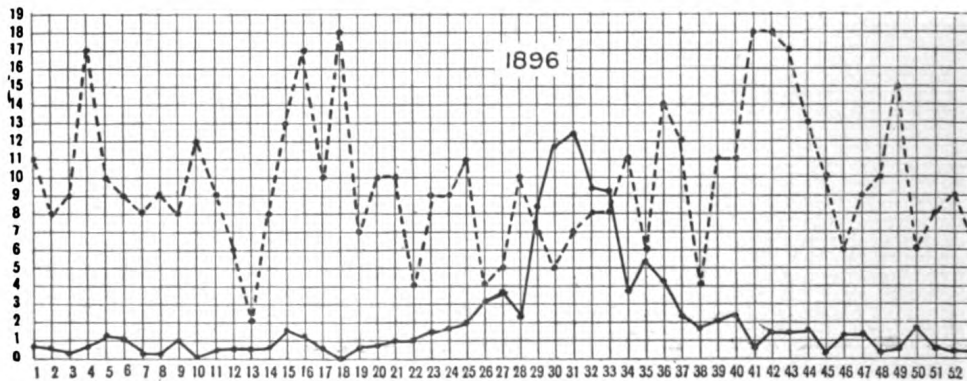
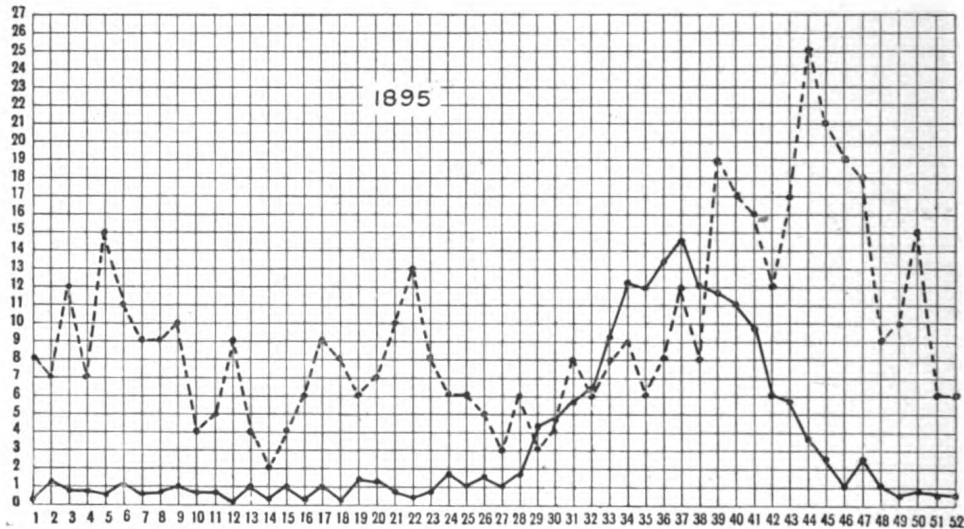


line marking the corresponding week of the year. Deaths from diarrhœa are divided by five, in order to render the curves more manageable, and the corresponding numbers are entered as black dots. This has the effect of flattening the diarrhœa curve, and to some extent obscuring its great

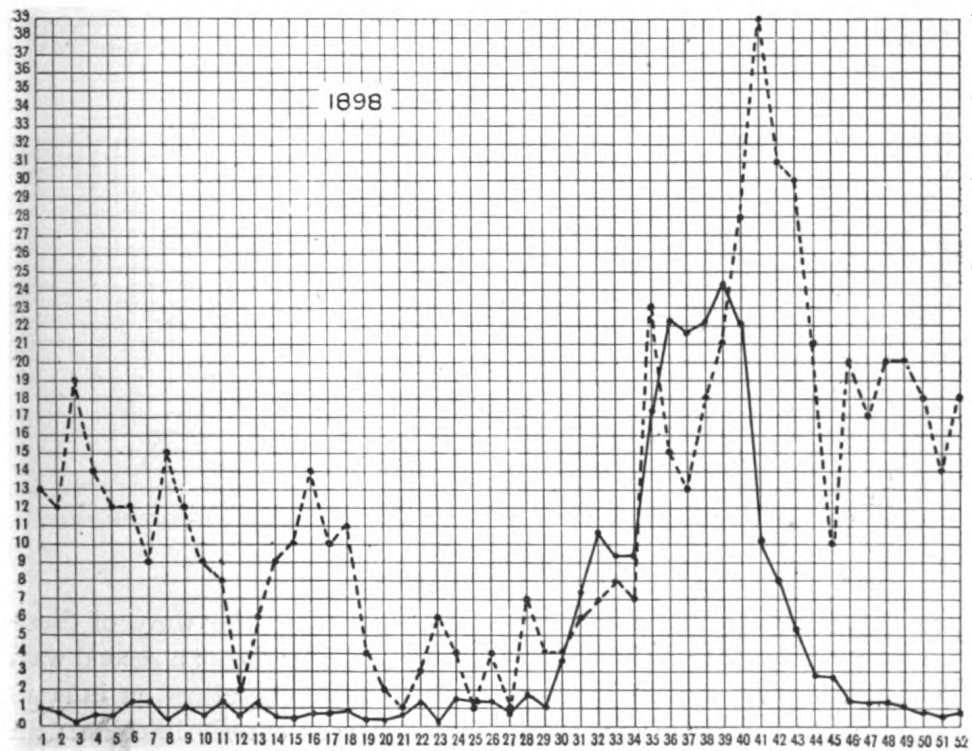
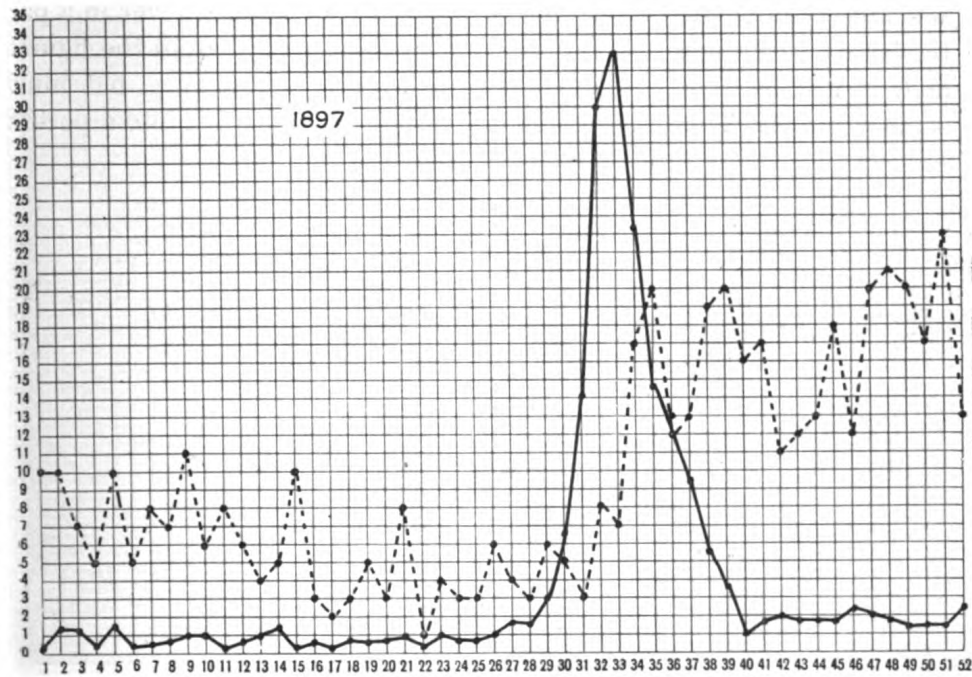


height. The imagination must therefore be here brought into play. The curve of diarrhœa cases will be something like a hundred times as steep as that here shown. In general, we perceive that a high diarrhœa

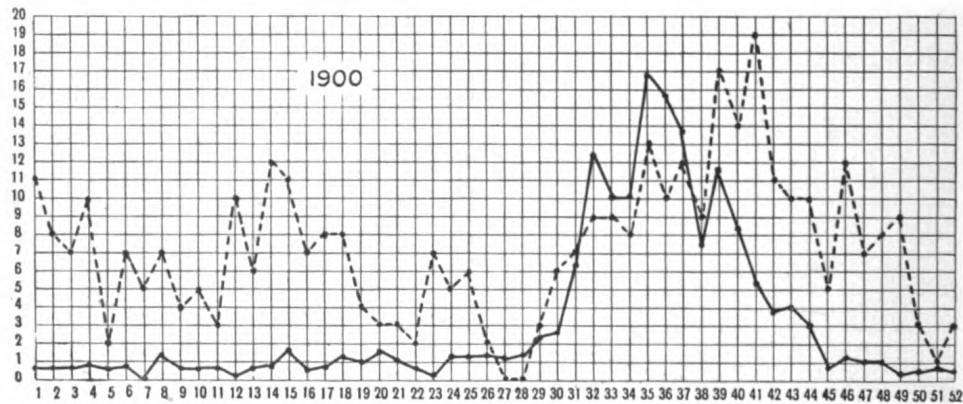
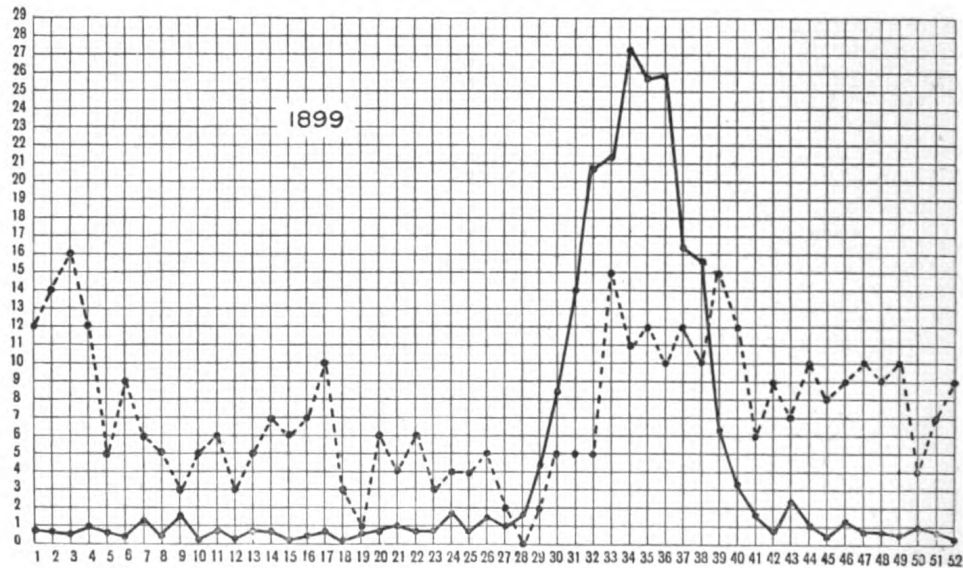
curve is accompanied by a high enteric curve, and an extended diarrhœa wave by an extended enteric wave. The enteric rises are much more considerable, relative to the diarrhœal, in the earlier than they are in the later years. They also begin earlier, relative to the diarrhœal curve.



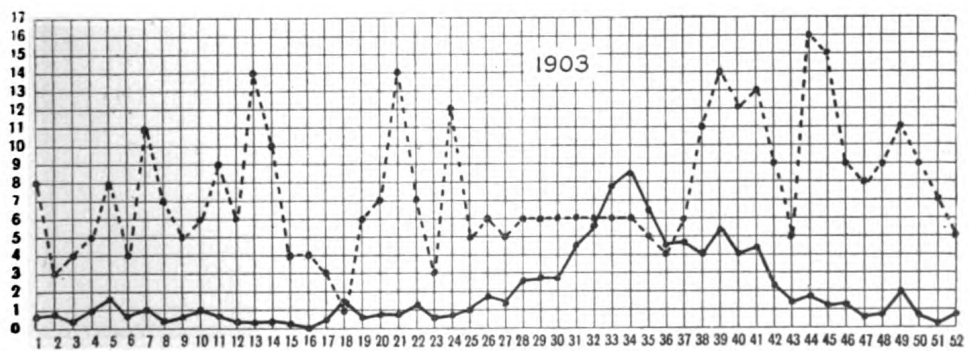
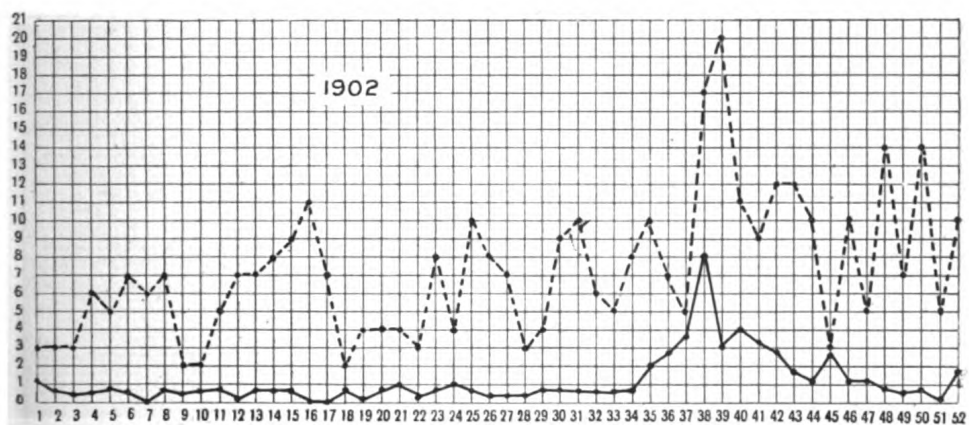
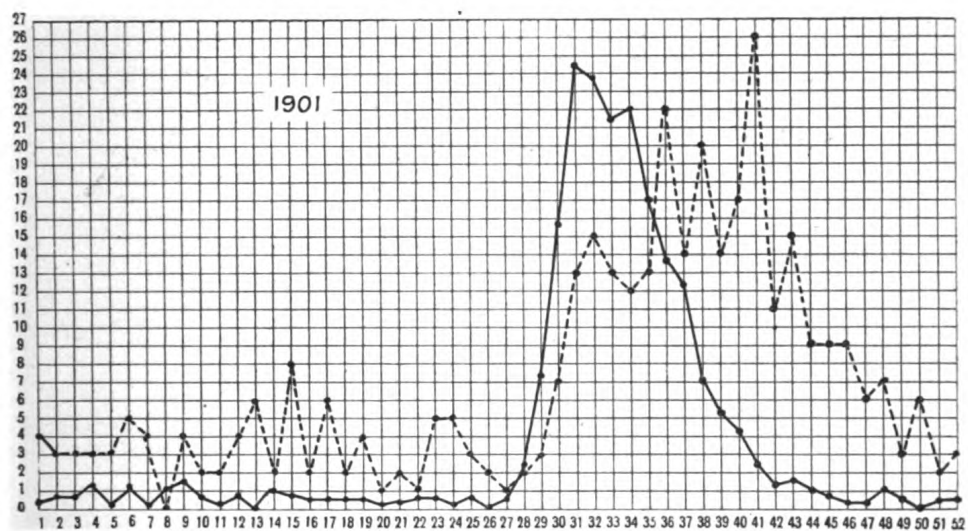
This cannot be connected with any condition of soil or temperature, the more so that the earlier years were years of low mean temperature in the third quarter, but may be explained by the greater prevalence of enteric fever in the earlier years, when a much smaller number of flies



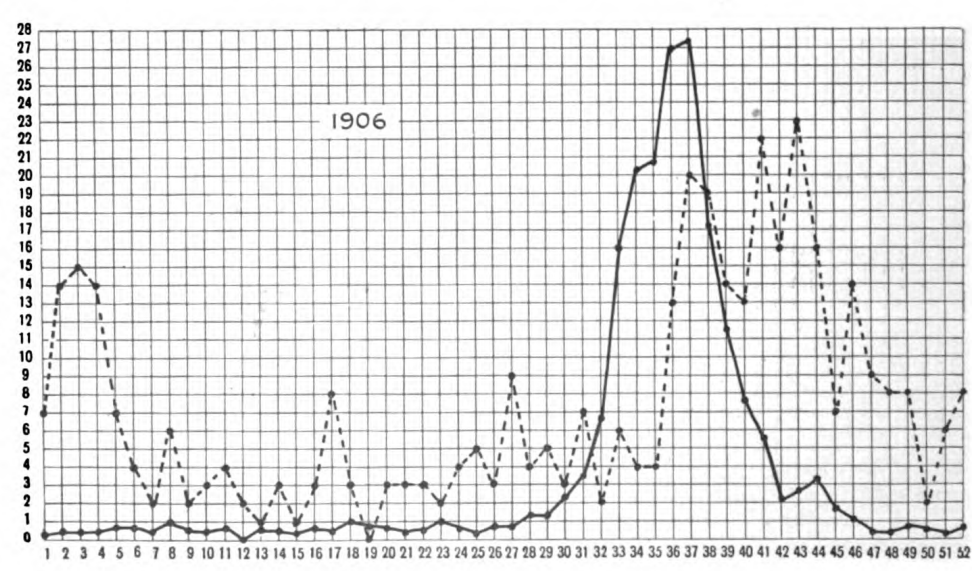
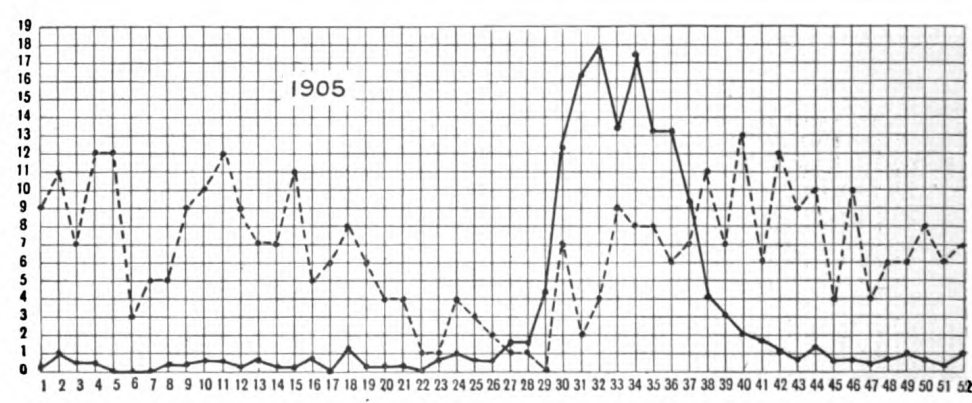
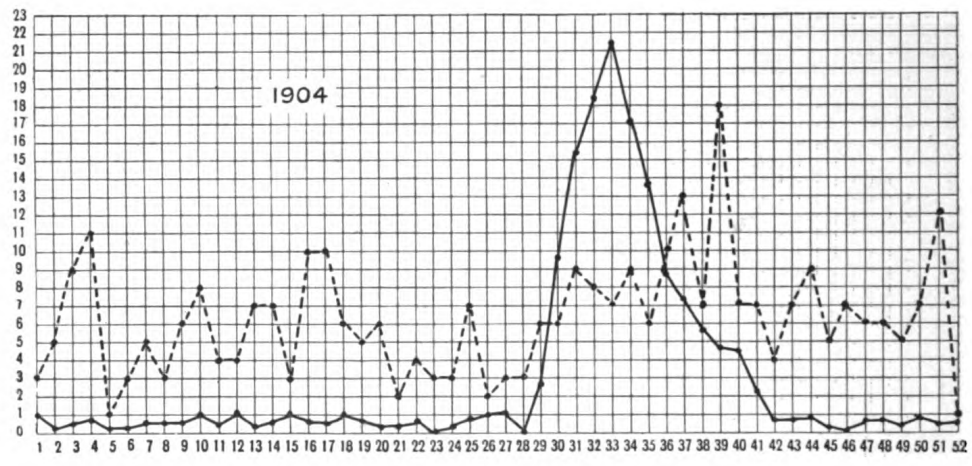
was able to raise the curve. The critical rise of enteric fever in a particular week is much less conspicuous in the curve than in the figures, and, it will be seen, is often attended with a preliminary but much smaller ascent. The autumnal enteric rise is often divided into a portion



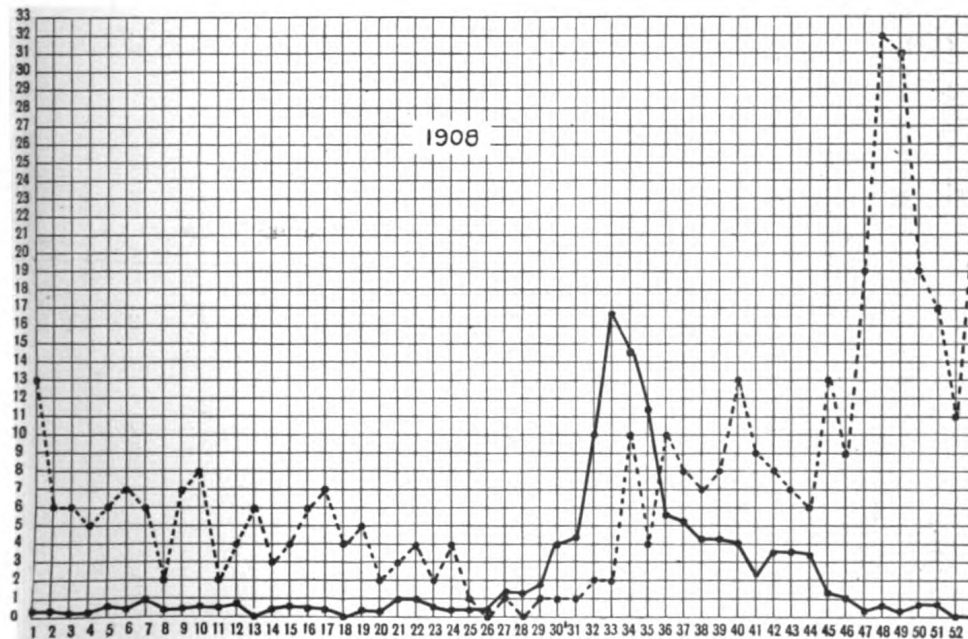
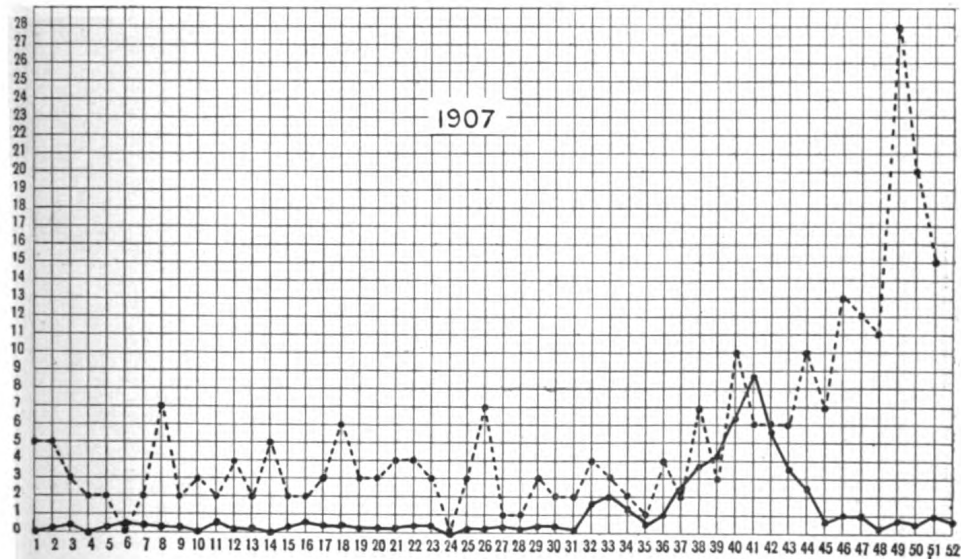
corresponding to the diarrhœal wave—that is, to the wave of flies—about ten days before the wave of deaths from diarrhœa; a second wave, which we have seen to be partly due to flies, operating on infection from overlooked cases, and on the now increased enteric infection; and



A-11a



even a third wave when the diarrhoea curve is much prolonged. These secondary and tertiary waves are in part produced by shellfish, especially towards the end of September and through October. We may exhibit the facts of separate years in the table on pp. 200 to 204.



FACTS RELATING TO THE CURVES OF ENTERIC FEVER.

Year	Maximum weeks of flies as determined from the meteorological data and from the course of diarrhoea deaths	Weeks of maxima of diarrhoea deaths, &c.	The so-called critical rise in enteric cases commencing	Weeks of ascent of the enteric wave	Features of the year
1891	Maximal weeks, 34 and 35; minor maxima, 31 and 32	Maxima, 36 and 37; minor maxima, 33 and 34; diarrhoea curve ascends 32 to 36, extends from 32 to 42; diarrhoea incidence low	34	Curve ascends, 32, 33, 34; primary wave, 32 to 39; secondary, 39 to 43; tertiary, 43 to 48; apex of primary wave precedes apex of diarrhoea curve by a fortnight	Primary wave due chiefly to direct influence of flies; secondary partly due to flies directly, partly to infection from overlooked cases, partly to mussels; tertiary wave mainly due to infection from overlooked attacks; mussels in evidence, weeks 1 to 10; nest or nests of infection, 11 to 19
1892	Maxima, 33 and 34	Maxima, 34 and 35; curve extends 31 to 40; diarrhoea incidence not severe	35	35; primary wave 35 to 41, secondary 41 to 49; 1	Preliminary slight rise in the enteric curve 29 to 34 probably due to flies; the primary wave partly due to infection from overlooked cases; the secondary wave more so; from weeks 1 to 11 the curve indicates much direct infection; from 34 to 41 mussels are in evidence
1893	27 (and 28?) secondary maxima 33 and 34	29, and minor maxima in 35; the curve of deaths extends from the 24th to the 42nd week; incidence severe	None distinct; the curve rises by a series of steps	The curve begins to rise in the 23rd week and ascends to the 31st week; there is a secondary increase in flies, and a fresh wave takes its start on the top of the previous one, 33 to 39; the tertiary wave extends from the 41st to the 44th week	The primary and secondary waves are merged 31 to 39; a tertiary wave is added 41 to 44, due in part to infection from overlooked cases; an extended entire enteric wave results corresponding to an extended entire diarrhoea wave; enteric wave large
1894	30 and 31	33; wave extends from 26th to 40th and from 40th to 44th weeks; incidence low	31	Primary curve 30 to 37; secondary 37 to 50	The primary curve of enteric corresponds to the main diarrhoea wave; the secondary wave is not high, and is probably due mainly to direct infection from previous cases, overlooked and otherwise; total incidence moderate

1895	32, 33, 34, 35, 36	37; flat, broad-shouldered top; considerable incidence	37 and 39	The enteric curve ascends in steps; the primary wave from 31 to 42; the secondary from 42 to 43	The long primary wave passes at its highest point into the mussel rise; up to the 38th week it corresponds well with the diarrhoea curve; the second step of the primary wave takes both direct infection by flies and indirect infection from overlooked cases; the secondary wave is largely due to the latter cause, but also to flies: from 4 to 9 and from 14 to 26 we have extension of local infection; considerable curve
1896	First maxima 28, 29, 30, 31; small rise 33	30 and 31; diarrhoea wave, 29 to 40; main portion. 28 to 34; incidence moderate to low	No critical rise	Primary wave slight, 31 to 38; secondary wave considerable and broad, 38 to 46; tertiary small wave 46 to 50	The secondary wave is large only by comparison with the slight primary wave; mussel infection appears to be indicated in the 4th, 16th, 18th, and 49th weeks
1897	31 and 32	33 (32); diarrhoea severe, sharp peak	34; primary enteric curve peaked; secondary curve prolonged and sustained	Primary wave, 32 to 36; this enteric wave bears a striking resemblance to the diarrhoea curve of deaths except in its termination; secondary wave 36 to 42; interval, tertiary wave 46 to 52	It looks as if the secondary wave terminates the influence of the fly period; if so, we may have to look for something special to explain the tertiary wave; the earlier features of the curve suggest mussel infection, but slight sustained high enteric
1898	Two maxima 34 and 35, 37 and 38	Prolonged maximum, 36 to 40; incidence very severe	35	Primary wave rises at first by steps, 31 to 45; this wave has two peaks, 35 and 41, corresponding to the two fly maxima; there is a correspondence generally with the diarrhoea curve; secondary wave, 45 to 52	The effects of a secondary rise of flies, coupled with infection from cases overlooked in the first part of the curve, result in a large outburst; the secondary wave is due to infection from overlooked cases? Incidence of enteric severe
1899	32, 33, 34, 35	34 to 36; the diarrhoea curve ascends steeply, has a flat top, and descends steeply, 29 to 41; incidence severe	33	33; the primary wave lasts from 32 to 41; it has a flat top from 38 to 39; the secondary wave is smaller than the primary	The secondary wave of 1898 continues up to the fifth week of this year. A minor wave 12 to 18 is due probably to some extension of direct infection, possibly to some extent to watercress. Incidence of enteric not severe in comparison of diarrhoea, due to failure of the secondary wave

Year	Maximum weeks of flies as determined from the meteorological data and from the course of diarrhœa deaths	Weeks of maxima of diarrhœa deaths, &c.	The so-called critical rise in enteric cases commencing	Weeks of ascent of the enteric wave	Features of the year
1900	Smaller maxima in 30, 31, a larger maxima 33, 34; the second maxima show no relation to the four-foot temperature	32 and 35, 36; incidence moderate; curve of diarrhœa, 29 to 44	35 ?	31 to 33; the primary wave lasts from 30 to 45, corresponding to the wide diarrhœa curve; it contains three successive ascents corresponding to the two ascents of the diarrhœa curve and the superposed secondary inflections. The secondary wave is small	There is a general correspondence between the primary enteric wave and the diarrhœa curve. The successive reinforcements of infection have time to come into play during the fly season. The secondary wave of 1899 lasts to the fifth week of this year; rises of the curve occur weeks 11 to 18, 22 to 25, due probably to extensions of contact infection, possibly to some extent to watercress; incidence moderate
1901	20, 30, 31, 32	31, 32, 33, 34: steep ascent and descent, rounded peak; incidence severe; curve 29 to 42	31	Ascends 30, 31, 32; primary wave blended with secondary to make one wave; 29 to 46	The primary wave shows a peak corresponding to that of the fly curve, then a superposed portion due to the descending fly curve, which rises above the first part of the wave; its character is observed by the zigzagging of the lines, indicating influence of mussels. Incidence of enteric fairly severe
1902	36	38; death-rate slight; extent of curve, 34 to 48	38	38, 39; primary wave, 37 to 41; secondary, 41 to 44; the later part of the curve zigzags, possibly due to influence of mussels	The enteric primary wave is high in proportion to diarrhœa, but closely agrees with it in time and character; the secondary wave is largely due probably to secondary infection from overlooked cases; the earlier portions on the curve suggest several extensions of local or direct infection, 8 to 9, 10 to 18, 24 to 27, 29 to 32
1903	31, 32; smaller second maximum, 38	33, 34; death-rate small; extent of curve, 28 to 42	38	37, 38, 39; primary wave absent; second wave, 37 to 43; secondary wave, 43 to 46	The enteric wave corresponding to the main diarrhœa wave is absent; there is, however, a primary wave corresponding partly to the secondary rise of diarrhœa, partly to the overlooked cases in the first part; there is also a secondary wave corresponding to this portion of the enteric curve; the behaviour of the earlier part of the curve suggests shellfish; these, however, do not, materially affect the wave, though they lower the rise in the 13th week

1904	Observed mum, 31	maxi- 33 (32 to 34) ; curve peaked ; curve, 29 to 41 ; incidence of diarrhoea severe	31 ?	31 : the primary wave coincides very nearly with the diarrhoea wave. There is, as usual, a secondary rise, corresponding to the descent of the diarrhoea curve ; no secondary wave follows this portion ; curve extends	There is, perhaps, no real distinction between what are now being called part of the primary wave and what in the earlier years were called secondary waves. They appeared, however, in the earlier years more completely separated off. The later part of the primary wave in 1904 and the secondary wave in 1891 are alike connected with the declining part of the fly curve, and indicate infection from an increased number of centres, as well as from overlooked cases arising at the height of the diarrhoea season ; enteric incidence small ; the peaks in the 37th and 39th weeks are due to mussels
1905	Observed mum, 31	maxi- 32 and 34 ; the diarrhoea wave extends from 27 to 41, and has a rounded top ; incidence severe	33	33 : the primary wave extends from 33 to 41 ; a secondary wave, from 41 to 44 ; both are small ; the earlier part of the curve shows a rise 9 to 16, due no doubt to extension by direct infection	The second part of the primary wave corresponding to the declining curve of flies zigzags, and suggests mussels ; the secondary curve suggests secondary direct infection ; the peak in the 46th week suggests mussels ; as a matter of fact the peaks in the 38th, 40th and 42nd weeks are due to mussels, while that in the 46th week is partly due to that cause of infection ; the main features of the curve are not otherwise altered ; incidence of enteric slight
1906	Observed ma, 34, 35	maxi- 35, 36 ; very severe	36	36, 37 : primary wave, 35 to 40 ; secondary wave, 40 to 45 ; tertiary wave, 45 to 50 ; primary and secondary waves high ; tertiary wave small	The primary wave closely resembles the diarrhoea wave ; the secondary wave really corresponds to the second part of the primary wave in years of less acute rise ; it marks possibly the action of a diminished number of flies on the increased number of enteric cases ; there is, however, usually a gap which suggests difficulties here ; the tertiary wave arises, no doubt, from this secondary wave ; the zigzags in the secondary wave suggest mussels ; incidence tolerably severe

Year	Maximum weeks of flies as determined from the meteorological data and from the course of diarrhoea deaths	Weeks of maxima of diarrhoea deaths, &c.	The so-called critical rise in enteric cases commencing	Weeks of ascent of the enteric wave	Features of the year
1907	Presumed maximum, 39	41; incidence not severe	Not clear; its place probably taken by a mussel increase	Slight primary wave, 39 to 46; no clearly marked secondary wave	Subsequent rise at the end of the year probably due to mussels
1908	Corrected maximum, 32; diarrhoea moderately severe; curve extends from 27 to 45	33 (34)	34	34; primary wave, 33 to 44; great secondary wave, 46 to 52	The primary part of the enteric rise which may be taken to terminate in the 38th week; zigzags; the secondary part, 38 to 44, is clearer; part of the secondary wave is due to mussels; the numbers traced to mussels in the last five weeks are 4, 6, 9, 12, 8; this does alter the character of the peak, but does not much lower its height; explanation (?); possibly it is due to an outburst of direct infection

The total effect of these curves is to produce a strong conviction that, allowing for differences in the disease, the curves of enteric fever represent the action of flies as plainly as do those of diarrhœa, although the result is comparatively slight. Supposing an uprush of enteric fever to occur in the primary wave, and supposing a considerable number of flies still in evidence, there is strong reason why a secondary wave should arise. The cases in the primary uprush will not become infective for two or three weeks.

Some time is lost, as a rule, in transmission, and the secondary wave will therefore not be developed until five or six weeks after the first. Further, a gap will usually arise between the primary and secondary wave, or perhaps one should say between the primary and secondary rises of the fly curve. This will be the more marked the more acute and the shorter in duration the primary curve has been. If we take the average period between infection and death in diarrhœa as twelve days, the enteric curve of cases should, in so far as it is due to the same part of the fly wave, either coincide with the diarrhœa curve of deaths or follow it at an interval of a week. If, now we regard the enteric fly-wave as consisting of a primary and secondary rise, the secondary part separated from the primary for the reasons given, we note that the entire enteric wave of cases commencing nearly coincides in point of time with the entire wave of diarrhœa deaths, with those slight but important modifications from 1891 to 1908 to which I have already alluded. In the earlier years this wave extends on both sides beyond the diarrhœa curve, while in the later it corresponds almost exactly. In 1896 the primary and secondary rises are, for some reason, both slight and indistinct. In 1903 the primary rise is doubtful, but the secondary is well marked. As we have seen, however, when studying the behaviour of enteric fever in districts, as shown on spot charts, the number of districts which take no part in the autumnal rise is very variable. The total phenomenon, though distinct, is small, and we must not therefore be surprised if occasionally it fails.

The difference in behaviour between the diarrhœa and enteric rises may be expressed in other words by repeating what has already been said, that the turnover of the diarrhœa infection as operated by flies is much more rapid than that of diarrhœal infection. Thus, if we take into account the tendency of enteric fever to occur in infective nests, the steady accession of mussel infection from the early weeks of September to the end of April, and the accession derived annually from flies, we might rest content that we have fairly explained the enteric curve.

The alteration in position and magnitude of the enteric autumnal rise between 1891 and 1908 is explained when we assume that a much smaller number of flies could produce the same result in earlier years, there being much more infective matter for them to visit, and no other explanation seems to fit. If this be true, then the connexion between flies and diarrhœa receives support from an unexpected quarter. But the support is even stronger than at first it seems, since with a sufficient number of flies, and easy access to near infectious stools, there would appear to be no doubt that flies do convey enteric fever. It follows that the close correspondence between flies and diarrhœa is almost certainly one of cause and effect.

It will be seen that the evidence from other sources connecting flies and enteric fever is an important part of this argument, and to this I will briefly recur. But I would first consider the curves a little longer, asking the question: Do the facts which I have collected tell the whole story? Is there nothing else, no residual phenomena of importance which are being neglected?

Before considering the evidence in favour of flies being the carriers of enteric fever we may ask whether the facts hitherto elicited suffice fully to explain the curve of enteric fever. There are various features of those curves which throw some doubt on this. Of these perhaps the most striking are the waves occurring late in 1907 and 1908. The most satisfactory way to settle this question would be to take those years in which individual cases have been most fully investigated and see whether there is a residuum of cases occurring in rises for which we have no explanation. When we do this for 1904 and 1905, accepting only in respect of direct infection cases in which the contact appears fairly sufficient, we obtain much reduced curves, which nevertheless show the same general features.

In 1904 the rise in the third and fourth weeks remains. The curve rises in the tenth week to the fourteenth, and again in the sixteenth to the twentieth. The ascent in the thirty-first week and the portion which we have ascribed to flies extending with a break to the forty-first week is clearly marked. Further small rises occur in the forty-third to the forty-seventh weeks, and again in the fiftieth and fifty-first weeks.

In 1905 the rise in the first to the fifth weeks remains as a rise from the second to the fourth. The swell in the curve from the seventh to the twenty-first week is, however, almost obliterated. The fly wave from the thirty-third to the thirty-seventh week remains. There is then, however, a dip of three weeks followed by a second rise from the

fortieth to the forty-fourth weeks. The small rise from the forty-ninth to the fifty-second week is well maintained.

The facts for 1904, 1905, and 1907 may be presented thus. The last year I owe to Dr. J. R. Hutchinson, who has given much study to the question of carrier cases of enteric fever.

1904.

Cases commencing in weeks, with, first those connected with shellfish, then those who have contracted the disease after contact with previous cases, shellfish and contact cases being successively subtracted.

	Jan. 9	Jan. 16	Jan. 23	Jan. 30	Feb. 6	Feb. 13	Feb. 20	Feb. 27	Mar. 5	Mar. 12	Mar. 19	Mar. 26	April 2
All cases ...	3	5	9	11	1	3	5	3	6	8	4	4	7
Shellfish cases ...	0	3	1	3	1	0	1	0	2	1	1	1	2
Less shellfish cases ...	3	2	8	8	0	3	4	3	4	7	3	3	5
Contact cases ...	2	2	0	2	0	0	1	1	2	2	1	0	0
Less shellfish and con- tact cases	1	0	8	6	0	3	3	2	2	5	2	3	5

	April 9	April 16	April 23	April 30	May 7	May 14	May 21	May 28	June 4	June 11	June 18	June 25	July 2
All cases ...	7	3	10	10	6	5	6	2	4	3	3	7	2
Shellfish cases ...	1	0	1	1	0	0	0	0	1	1	0	0	0
Less shellfish cases ...	6	3	9	9	6	5	6	2	3	2	3	7	2
Contact cases ...	0	1	1	1	2	0	1	2	0	2	1	2	0
Less shellfish and con- tact cases	6	2	8	8	4	5	5	0	3	0	2	5	2

	July 9	July 16	July 23	July 30	Aug. 6	Aug. 13	Aug. 20	Aug. 27	Sept. 3	Sept. 10	Sept. 17	Sept. 24	Oct. 1
All cases ...	3	3	6	6	9	8	7	9	6	10	13	7	18
Shellfish cases ...	0	0	0	0	0	0	1	0	0	0	1	1	6
Less shellfish cases ...	3	3	6	6	9	8	6	9	6	10	12	6	12
Contact cases ...	0	0	3	2	0	3	0	1	3	2	5	1	1
Less shellfish and con- tact cases	3	3	3	4	9	5	6	8	3	8	7	5	11

	Oct. 8	Oct. 15	Oct. 22	Oct. 29	Nov. 5	Nov. 12	Nov. 19	Nov. 26	Dec. 3	Dec. 10	Dec. 17	Dec. 24	Dec. 31
All cases ...	7	7	4	7	9	5	7	6	6	5	7	12	1
Shellfish cases ...	2	0	0	2	3	0	1	4	1	1	2	0	0
Less shellfish cases ...	5	7	4	5	6	5	6	2	5	4	5	12	1
Contact cases ...	1	3	4	0	4	0	3	1	5	2	0	1	0
Less shellfish and con- tact cases	4	4	0	5	2	5	3	1	0	2	5	11	1

1905.

Cases of Enteric Fever notified in 1905, arranged according to the numbers commencing week by week ; those traced with definiteness to contact with previous cases, and those associated with the previous consumption of shellfish, nearly always mussels, being similarly arranged, and then deducted. Doubtful cases are here not excluded.

	Jan. 7	Jan. 14	Jan. 21	Jan. 28	Feb. 4	Feb. 11	Feb. 18	Feb. 25	Mar. 4	Mar. 11	Mar. 18	Mar. 25	April 1
All cases commencing	8	6	5	8	12	4	2	4	12	7	13	10	5
Cases traced to contact	4	1	3	1	4	2	0	3	2	4	5	6	3
Associated with mussels	3	3	0	2	2	2	0	1	5	2	3	1	2
The last two deducted	1	2	2	5	6	0	2	0	5	1	5	3	0

	April 8	April 15	April 22	April 29	May 6	May 13	May 20	May 27	June 3	June 10	June 17	June 24	July 1
All cases commencing	10	8	4	6	6	6	3	3	1	1	2	3	2
Cases traced to contact	1	0	0	0	4	4	0	1	1	1	0	0	1
Associated with mussels	6	3	2	6	1	2	0	0	0	0	0	0	0
The last two deducted	3	5	2	0	1	0	3	2	0	0	2	3	1

	July 8	July 15	July 22	July 29	Aug. 5	Aug. 12	Aug. 19	Aug. 26	Sept. 2	Sept. 9	Sept. 16	Sept. 23	Sept. 30
All cases commencing	1	0	1	6	2	5	8	6	5	3	8	12	10
Cases traced to contact	0	0	0	1	0	0	0	1	1	2	0	1	4
Associated with mussels	1	0	1	1	0	1	1	2	1	1	4	4	3
The last two deducted	0	0	0	4	2	4	7	3	3	0	4	7	3

	Oct. 7	Oct. 14	Oct. 21	Oct. 28	Nov. 4	Nov. 11	Nov. 18	Nov. 25	Dec. 2	Dec. 9	Dec. 16	Dec. 23	Dec. 30
All cases commencing	10	6	14	7	11	7	5	3	9	5	8	6	6
Cases traced to contact	1	1	3	0	2	0	0	1	2	1	0	0	0
Associated with mussels	5	2	6	3	4	2	2	2	3	0	2	0	0
The last two deducted	4	3	5	4	5	5	3	0	4	4	6	6	6

1907.

Cases of Enteric Fever commencing in the week ending :—

	Jan. 5	Jan. 12	Jan. 19	Jan. 26	Feb. 3	Feb. 10	Feb. 17	Feb. 24	Mar. 2	Mar. 9	Mar. 16	Mar. 23	Mar. 30
All cases ...	3	4	5	2	1	3	0	4	3	2	4	4	3
Traced to contact infection	0	0	0	0	0	0	0	2	1	0	0	1	1
Connected with shellfish	1	2	1	0	0	0	0	0	0	0	1	1	0
Less contact and shellfish cases	2	2	4	2	1	3	0	2	2	2	3	2	2
	Apr. 6	Apr. 13	Apr. 20	Apr. 27	May 4	May 11	May 18	May 25	June 1	June 8	June 15	June 22	June 29
All cases ...	2	5	2	2	4	5	2	3	3	5	3	1	4
Traced to contact infection	0	0	0	0	1	1	0	0	0	0	0	0	0
Connected with shellfish	0	0	0	2	1	2	1	1	1	2	2	1	2
Less contact and shellfish cases	2	5	2	0	2	2	1	2	2	3	1	0	2

	July 6	July 13	July 20	July 27	Aug. 3	Aug. 10	Aug. 17	Aug. 24	Aug. 31	Sept. 7	Sept. 14	Sept. 21	Sept. 28
All cases ...	7	0	0	1	2	4	2	2	2	1	4	3	5
Traced to contact infection	3	0	0	0	1	1	2	1	0	0	0	0	2
Connected with shellfish	1	0	0	0	0	0	0	0	1	1	1	1	2
Less contact and shellfish cases	3	0	0	1	1	3	0	1	1	0	3	2	1

	Oct. 5	Oct. 12	Oct. 19	Oct. 26	Nov. 2	Nov. 9	Nov. 16	Nov. 23	Nov. 30	Dec. 7	Dec. 14	Dec. 21	Dec. 28
All cases ...	4	10	6	6	8	12	8	13	15	16	16	19	16
Traced to contact infection	0	0	0	0	0	4	1	3	1	1	1	1	4
Connected with shellfish	1	6	3	2	3	6	4	3	6	7	7	5	5
Less contact and shellfish cases	3	4	3	4	5	2	3	7	8	8	8	13	7

The effect of these repartitions of cases is somewhat to heighten the impression that there may be something unexplained. Now, this something might be persistent typhoid carriers, although this is unlikely. It is unlikely, I mean, that there should exist the amount of infection from this cause able to cause these disturbances. There is also the possibility that we have not exhausted the modes or means of conveying enteric fever. Not to speak of food infections from overlooked cases, infected closets from the same cause, carriage in infected clothing, there may be other living carriers besides flies. It is, however, difficult to see how these are to operate in winter except along adjoining houses. The living agents known to us are fleas, bugs, cockroaches, mice, and rats. Fleas and bugs we may put aside. Enteric is not found to any extent in their special haunts. Cockroaches deserve some attention. Like flies, they occur in immense numbers, and they are very often present in the poorer sort of houses, especially, I think, near stables. They are in Manchester widely distributed. That they visit beds I have positive knowledge. They often swarm about the kitchen at night, and investigate whatever food is left about. It is not difficult to ascertain whether they have visited food, on account of the smell which they leave. I have no proof, however, that they cause spread of enteric fever. The facts about them which I have been able to collect are these: They exist often in unsuspected numbers amongst cinders. A street was paved with cinders, and the covering of it with stone setts was accompanied by the migration of large numbers of cockroaches into the houses with simultaneous appearance of mice. It is probable that in warm, dry weather they are to be found in considerable numbers in middens. I have been informed by a gentleman who is interested in insects

that they certainly do visit middens. They are known to visit closets. Cockroaches have been seen to move towards the house in considerable numbers at dusk, but my informant could not say at what period of the year this occurred. I was informed by a stableman that cockroaches were found in the manure stead in summer in large numbers, and occasionally in winter. This manure stead was at the end of a row of houses, in many of which I ascertained that they occurred in large numbers. I have visited two rows of houses in which cockroaches are numerous. In both, mice occur in considerable numbers. It is possible that mice feed on them. Rats are said to eat them. They are said to be most numerous in the houses in wet weather. They are caught in the "Demon" trap, or killed by poison. They breed slowly, so that they do not return for a considerable time after they have been thinned down; but the young ones come out. One householder informed me that a little whisky put in the cup of the "Demon" trap increased the numbers caught considerably. Flies also appear partial to alcohol. Notwithstanding that they are partial to stables, it is doubtful whether the animals observed in the manure stead were not the beetles which feed on the larvæ of flies, not cockroaches. I also ascertained that many blackbeetles were found in summer in another heap of house manure; but here it was even more doubtful whether they were cockroaches. We have seen that flies also probably live in hollow places in the walls in winter. It is said that the ordinary blatta has no antagonism to flies. The larger blatta, *Blatta gigantea*, is stated in Lardner's Encyclopædia to devour flies. It is possible that the ordinary cockroach is not so innocent in respect of flies as it is believed to be. Now, the rat virus is a species of coli bacillus, and it is just possible that they get this virus naturally from eating cockroaches. Whether there is exchange of any coli virus between these animals or not, it is at all events possible that if present in large numbers they aid in disseminating infection. Rats, of course, usually have access to sewers, and may thus carry infection on their feet. There appears to be a great antagonism between rats and mice. Probably rats kill mice, and eat them. There must be some plagues which destroy both cockroaches and mice, unless they die of sheer inanition. Do beetles sustain the *Empusa muscæ* from season to season? It seems to me that the occurrence and movements of cockroaches, their relations to flies, to each other, and to the occurrence of multiple cases, require to be worked out, even if it be to be put aside. In particular it is desirable to know their movements in summer and winter respectively. The movements of mice and rats appear particularly worthy of study.

This may, at first sight, seem a vain suggestion. Many of our histories of contact infection, however, are unsatisfactory in point of time or in precision as to the mode of conveyance. The occurrence of enteric fever in districts in nests, illustrated by the experience of St. George's Ward in 1905, is in point. The occurrence of these local nests is not confined to the fly season. It may be that the movements of some of our domestic pests will account for the establishment of these nests. They are, however, not likely to be due to cockroaches, which would be more likely to account for the spread of the disease in one family, and from that family to visiting acquaintances. Further, the local nests of enteric are most apt to occur in warm weather, when, of course, they may be due to the movements of flies. At all events, the matter appears worth investigating.

This discussion may be thus summed up: In Manchester, with sufficient energy and skill, the probable origin of most cases of enteric fever can be determined, in some years at all events, in terms of mussels and "contact" infection. "Contact" infection and "mussel" cases are proportionately least numerous prior to the thirty-eighth week of the year, before which period the enteric fever curve begins to ascend. This ascent, which cannot be due to either of the two causes for the continuance of enteric fever which have been named, may be due to transference of infection by flies. When the number of cases of enteric fever, commencing week by week, and the number of deaths from diarrhoea occurring in the same week are plotted out in curves, it will be seen that a rise in the enteric curve occurs corresponding to the summer wave of diarrhoea deaths. This is usually broken up into primary and secondary rises, the primary rise coinciding with the main curve of flies, but coming about a fortnight later. The secondary rise is often larger than the first, and is interpreted as being due to transference by a smaller number of flies of a much increased amount of infection. These waves were larger from 1891 to 1896 than they have been in recent years, notwithstanding that the number of flies was probably much smaller, and began earlier relative to the rise in diarrhoea deaths, although the corresponding temperatures were lower and less favourable to the development of enteric fever than they have since been. The effects of temperature on soil cannot explain this; on the other hand, the death-rate from enteric fever has fallen to less than one-third of its amount in the earlier period. The number of centres of infection of enteric fever was therefore formerly much larger than it now is, and a much smaller number of flies would thus be able to make an impression on the

curve. When diarrhœa is high, the enteric primary rise tends to approximate to the curve of diarrhœa deaths, and becomes well marked. These results can only be expressed in terms of a common cause, the difference in the waves depending on the long latent period of enteric fever and its late infectiveness. Enteric fever, however, at all times has a slight incidence in Manchester compared with diarrhœa, and its rises are therefore less marked and definite; so slight, in fact, is the disease that in some weeks no cases are reported. Occasionally, though rarely, the rise corresponding to the diarrhœal ascent is ill-marked, or remains out. Especially has this tendency been manifested in recent years. The rise of the curve of enteric cases has, from starting before the rise of diarrhœa deaths, been gradually moved onwards to coincide with the apex of that rise. These facts are most easily interpreted, it seems to me, in terms of flies operating on the much smaller number of infective centres which now exist.

When all three proved reinforcements of the enteric curve are allowed for, however—contact infection, mussels, and flies—there remain residual increases the causes of which deserve special attention.

If the main autumnal rise of enteric fever be due to flies, it almost follows that the correspondence between flies and diarrhœa is one of cause and effect, a result which we have already reached by a process of exclusion.

EXTERNAL EVIDENCE AS TO THE CONNEXION OF FLIES WITH THE CONVEYANCE OF ENTERIC FEVER.

The evidence connecting flies with the dissemination of enteric fever in tropical countries appears practically conclusive. Undoubtedly the most important single piece of work on this subject is that of the American Army Commission on the prevalence of enteric fever among soldiers in 1898. In 1898 practically every regiment developed enteric fever, which became epidemic in the encampments both of the Northern and Southern States. "Most, probably all, of the regular regiments developed enteric fever less than eight weeks after going into camp."¹ The spread of the disease was, in the opinion of the Commission, due to camp pollution. The number of cases varied with the method of disposing of the excreta. The tub system and regulation pits were unsatisfactory. Infected water was not an important factor. On one

¹ *Lancet*, 1900, i, p. 1916.

occasion part of the camp used a possibly contaminated water, the other a pure water. Both were alike affected. Only about half the cases were correctly diagnosed. Flies undoubtedly served as carriers of infection. It is probable, also, that the disease was disseminated by dust, though this is regarded as a minor factor.

In his very useful summary of the epidemiological evidence, Mr. Gordon Hewitt¹ quotes Dr. Vaughan, a member of the Commission, as follows: "My reasons for believing that flies were active in the dissemination of typhoid fever may be stated as follows: (a) Flies swarmed over fæcal matter in the pits, and then visited and fed upon the food prepared for the soldiers in their mess tents. In some instances, where lime had recently been sprinkled over the contents of the pits, flies with their feet whitened with lime were seen walking over the food. (b) Officers whose mess tents were protected by screens suffered proportionately less from typhoid fever than did those whose tents were not so protected. (c) Typhoid fever gradually disappeared in the fall of 1898 with the approach of cold weather, and the consequent disabling of the fly."

American opinion seems to run strongly in this direction, and we may refer to the remarkable facts adduced by Jackson as to the connexion between the polluted foreshore at New York and the prevalence of enteric fever in the neighbourhood, carried, as he believes, by flies.

Now the typhoid bacillus is not killed by cold, and, if it is merely a question of infection, our own experience shows that its incidence declines slowly in cold weather. But, of course, the seasonal impression made by flies in this country is comparatively small. On the other hand, we have plenty of dust at other periods of the year, notably in March and June, during which months, however, diarrhœa remains motionless, while June and July coincide with the nadir of enteric fever. It is not until after the commencement of the fly season that either diarrhœa or enteric fever begins to move.

In his Milroy lectures Dr. Waldo developed the thesis that dust, and in particular the dust derived from horse droppings, is responsible for the summer outbreak of diarrhœa. This idea of dust conveyance seems to be an obsession, and suitable rather for the facts of South Africa than for our conditions. It is again mentioned chiefly to draw attention to the very acute letter by Mr. W. Salisbury Sharpe in the *Lancet* of June 2, 1900, in which he develops the idea that flies are the real

¹ Gordon Hewitt: "The Structure, Development and Bionomics of the House Fly," *Quart. Journ. Micro. Sci.*, 1909, liv, p. 347.

carriers. There is so much matter in it that I should like to quote it in full. I must content myself with saying that it contains suggestions which even yet will be found valuable in the investigation of the disease. It may be noted, however, that he points out that diarrhœa prevails most in houses least accessible to the movement of dust, in which the windows of the living-room are kept closed and in which flies gather in large numbers. As regards Dr. Waldo's suggestion that horse manure may contain the specific infective matter of diarrhœa, this is disposed of in the same manner by the seasonal occurrence of diarrhœa which coincides with that of flies. There is, so far as I can ascertain, no seasonal affection of horses preceding the seasonal human diarrhœa to lend colour to the suggestion. There is, however, an interesting connexion between horses and enteric fever quoted by Mr. Gordon Hewitt from a communication by Aldridge to the Army Medical Department Report, 1902, p. 207 : " In the British Army in India, 1902-5, the ratios per 1,000 per annum of cases of enteric fever admitted into hospital were : Cavalry 41.1, and infantry 15.5 ; in the United States Army, cavalry 5.74, and infantry 4.75." The connexion is, no doubt, between horses and the number of flies.

Dr. Snell, of Coventry,¹ in 1906 showed that 70 per cent. of the cases of infantile diarrhœa occurred in the north-east part of his district, close to a large collection of refuse where flies swarmed. There is testimony to the like effect from medical men who were in the Boer campaign. Dr. Arnold H. Watkins, writing from Kimberley,² says : " With the advent of cold weather the flies disappear, so does almost entirely the typhoid." Dr. A. B. Dunne³ writes from Bloemfontein : " Nothing was more noticeable than the fall in the admissions from enteric fever coincident with the killing off of the flies on the advent of the cold nights in May and June." Dr. Howard H. Tooth⁴ is of opinion that too much stress has been laid on water supplies. At the Modder River, " devils," or storm dusts, swept through the camp, and would lift the contents of pits. This was not so much the case at Bloemfontein. He does not exclude the action of dust, but observes that at both places there were enormous numbers of flies. He also remarks : " The disappearance of enteric fever and flies with the first appearance of frost at night may be more than a coincidence." All these observers, then, fix

¹ Quoted by Ainsworth, *Journ. Roy. Army Med. Corps*, 1909, xii, p. 485.

² *Brit. Med. Journ.*, 1900, ii, p. 787.

³ *Brit. Med. Journ.*, 1902, i, p. 622.

⁴ *Brit. Med. Journ.*, 1900, ii, p. 1368.

on the simultaneous destruction of flies and diminution of enteric fever. The dust then becomes inoperative.

A very interesting paper by Dr. A. H. Ainsworth on "The House Fly as a Carrier of Disease" is given in the *Journal of the Royal Army Medical Corps*, May, 1909.¹ The enteric curve ascends during the heavy rains of the monsoon in Poona, and descends before their decline. This would seem absolutely to dispose of dust as a principal factor. The enteric curve, however, follows on the advent of flies, the curve of which also ascends and descends during the season of heavy rains; moisture is, of course, necessary for their development. There is no correspondence between the amount of rainfall and the amount of diarrhoea, although the prevalence of heavy rainfall coincides in time with the ascent in enteric fever and diarrhoea. The observations on flies relate only to one year, and are defective in amount; they require to be much extended. The author, however, considers flies to be the carriers of infection. This most interesting communication, illustrated as it is by a series of charts showing prevalence of rainfall, enteric fever, and, in one year, of diarrhoea, would seem to dispose entirely of the view that it is by dust that the ascent of these diseases is caused; while, though incomplete, it emphasizes the part played by flies in carrying these diseases. It may be said that the explanations of the observed facts have not all been to this effect. If, however, the statements quoted be correct we can interpret for ourselves, and the observations referred to appear to furnish a practically conclusive case for the potency of the house fly as a carrier of enteric (and diarrhoeal) infection at home as well as abroad.

We may now consider those conditions for the conveyance of the infections of enteric fever and diarrhoea which have not been fulfilled in the argument:—

(1) Are house flies present in immense numbers in houses prior to primary attacks in those of infants? In most cases they are. For details I must refer to my annual reports for 1904 and 1905. It is true that this is not always the case; but one does not maintain that every infant is infected through the agency of flies, though many are believed to be so indirectly who are not directly.

(2) Have house flies been shown to convey bacteria? They have. It is unnecessary on this point to multiply references. That they carry away enteric bacilli from enteric stools is shown by Major Firth and Major Horrocks in their communication to the *British Medical Journal*,

¹ *Journ. Roy. Army Med. Corps*, 1909, xii, p. 485.

1902, ii. An important contribution to this subject by Dr. Sellers, working in Professor Delépine's laboratory, is given in the annual report on the health of Manchester for 1906. The collection of the flies was made by a method devised by Professor Delépine, and designed to secure that the traps were free from any micro-organisms except those carried in by flies. The flies were captured at twenty-eight stations, such as to give a good chance that they should be carrying the infective germ of diarrhœa. The largest number taken in one trap was about twenty. As to the methods of examination I must refer to the article itself. Dr. Sellers states: "As regards the actual number of bacteria present, it was found that considerable variations occurred. Generally speaking, a quantity of washings corresponding to a single fly yielded hundreds or even thousands of colonies." Of the suspicious bacteria found there were five which clearly belonged to the *Bacillus coli* group. The total number of flies examined was 380.

It is probable from the above investigation that it requires the visit of a very large number of flies before infection can take place, even supposing that many of these come from infective excreta.

The other conditions laid down for the completion of the argument have, I hope, been fulfilled, and it will be for this Society to consider whether it has been established that no hypothesis other than that of transmission and of infection by flies will adequately explain the diarrhœa curve, or the primary autumnal disturbance of the enteric curve in Manchester.

Epidemiological Section.

April 22, 1910.

Dr. JAMES NIVEN, President of the Section, in the Chair.

Contribution to the Study of the Influences determining the Prevalence of Bovine Tuberculous Mastitis.

By SHERIDAN DELÉPINE, M.B.

(1) INTRODUCTION.

IN studying the mode of spread of any infectious disease it is always necessary to keep in mind the complexity of the conditions influencing or determining the occurrence of infection. To the pathologist an infectious disease is invariably the result of the action of an invading or parasitic organism upon an invaded organism or host.

The occurrence of a case of "tuberculosis vera" may be taken as absolute proof that Koch's bacillus has penetrated and multiplied in the tissues of an individual in whom it has caused the symptoms and lesions which we associate with tuberculosis. The converse is not true, and we cannot say that the presence of tubercle bacilli in a locality, or even within the body of an individual, is invariably followed by the production of tuberculosis. This sequence is probably the exception. The occurrence of cases of infectious disease is not determined only by the presence of the essential causal agent, but also by the co-operative agency of various factors, some of which become, under certain circumstances, so important that they more or less completely overshadow the essential cause. This is particularly noticeable in the case of diseases, such as tuberculosis, which are caused by widely-distributed microbes. If, whenever the opportunity of infection occurred, the bacillus were abundant or virulent enough, and its possible victims were in a receptive state, there would be very few persons free from tuberculosis among civilized communities.

The importance of predisposing factors is often so great that not a few clinicians and administrators have given but scant attention to the essential cause. It must be acknowledged that in many cases it is possible to obtain excellent results by dealing thoroughly with predisposing causes, and often there is no other course open to us. It is, however, never safe to disregard, even temporarily or partially, the *causa causans*, because it is a variable factor, and may, under the influence of various circumstances, acquire infective powers exceeding the average. Neither is it safe to trust entirely the action of natural or artificial curative agencies. Recoveries from infectious diseases may be apparent or real, and do not invariably result in so great a reduction in the number of infective centres as might be expected. Recoveries brought about by natural processes introduce also an additional element of complexity in our estimates of the incidence of various infectious diseases.

In studying the conditions which influence the prevalence of any infectious disease, we must therefore take account of the possible action of a great number of factors which may roughly be grouped into the following categories:—

Distribution and habits of the infecting parasite.

Conditions influencing the quantity of the parasite.

Conditions influencing the virulence of the parasite.

Opportunities and channels of infection.

Conditions influencing the resistance of the possible host.

Proportion and completeness of recoveries.

I have thought it necessary to make these few preliminary, and I might almost say commonplace, remarks to make it clear that I have no illusion regarding the completeness of the facts upon which this communication is based. I must also at this stage explain that it is not my intention to enter upon a discussion of the relations of bovine to human tuberculosis. I am fully convinced of the unity of human and bovine tuberculosis—a unity which does not preclude variations. I believe that, both in man and in cattle, infected food is an important conveyer of tuberculous infection, and that the milk of cows suffering from tuberculous mastitis is the most important means of transmission of bovine tuberculosis to the infant.

The relations between human and bovine tuberculosis were originally the main object of my investigation, but, having defined my position, I will not refer to the matter again, and will deal only with bovine tuberculosis. The subject which I have selected is, I believe, interesting

in itself, and may be looked upon as belonging to a branch of epidemiology which might be termed "experimental or comparative epidemiology."

(2) TUBERCULOSIS OF THE UDDER IN RELATION TO TUBERCULOSIS
OF OTHER ORGANS.

Cows affected with tuberculosis of the udder are, in the majority of cases, suffering also from more or less advanced tuberculosis of other organs. I have personally examined post-mortem the organs of a great number of cows affected with tuberculous mastitis, and in only one case out of nearly 100 have I failed to discover tuberculous lesions of internal organs. In three more cases out of the same number the internal lesions were limited; in the great majority of cases they were extensive. Out of more than 300 cows suffering from tuberculosis of the udder, and examined by several veterinary surgeons, only four have been reported to me as having been free from any other tuberculous lesions. Primary tuberculosis of the cow's udder would therefore appear to be of rare occurrence.

Tuberculous mastitis is not common in young cows. I estimate that about 90 per cent. of the animals suffering from this lesion are middle-aged or old cows—i.e. cows over four or five years of age. I regret not to be able to give more definite information on this point, which is not without importance; but, in the absence of any regular system of registration and inspection of cows, it is practically impossible for a veterinary surgeon to ascertain the exact age of all the cows inspected by him during an occasional visit to a farm. An experienced man can, however, estimate rapidly and fairly accurately whether a cow is young or old. It is mostly upon data gathered in this way, more especially by Mr. J. W. Brittlebank, whose experience in such matters is well known, that my opinion regarding the age of cows is based.

From personal observations made between the years 1896 and 1898 I came to the conclusion that in about 3·7 per cent. of all cows suffering from tuberculosis of any organ the udder was affected with tuberculosis.¹ This estimate was rather higher than those made at the time by three well-known veterinary authorities, but subsequent experience has shown me that even my estimate was barely high enough. It may therefore be safely assumed that for each cow affected with tuberculous mastitis there are on an average about twenty-seven cows with tuberculous

¹ "Prevention of Tuberculosis in Cattle," *Veterinarian*, Lond., 1899, lxxii, pp. 453, 528, 683.

lesions of other organs. From the facts collected in this brief summary it seems reasonable to infer:—

(1) That the presence of a tuberculous udder on a farm is in the great majority of cases evidence that there is on that farm at least one cow suffering with advanced, or fairly advanced, tuberculosis.

(2) That the number of cows found affected with tuberculosis of the udder in a district may be taken as a rough index of the probable number of tuberculous cows in that district.

A study of the conditions influencing the prevalence of tuberculous mastitis has therefore distinct bearing upon the whole problem of bovine tuberculosis. The importance of this conclusion depends on the facts: (1) that tuberculous mastitis can, by bacteriological methods, be diagnosed with great accuracy; (2) that no other accurate method is at present available for estimating approximately the relative prevalence of bovine tuberculosis in the various parts of any extensive area; and (3) that, through the work done in Manchester, we are now in possession of a large mass of reliable data regarding the incidence of tuberculous mastitis in a considerable area (nearly 5,000 square miles).

If systematic tuberculin testing of all cattle could be practised it would be easy, in a comparatively short time, to obtain more complete data, notwithstanding the fact that the tuberculin reaction often fails in old cattle suffering from advanced tuberculosis; but this simple method was not available fourteen years ago, when I began this investigation.

The testing of milk for the presence of tubercle bacilli, though it does not indicate directly the total number of tuberculous animals, has an advantage over the tuberculin test in that it reveals the cows that are the most dangerous to human health.

(3) COLLECTION OF DATA; EXAMINATION OF SAMPLES OF MILK; INSPECTION OF COWS AND SHEDS.

A short statement of the manner in which the Manchester Milk Clauses (1899) have been administered under Dr. Niven's direction during the last eleven years will make clear the nature of the evidence upon which my statistics are based.

Samples of cows' milk are taken by the food and drugs inspector from the milk-cans on their arrival at the railway stations or elsewhere within the city. Each milk-can contains the milk of several cows, and for this reason the milk obtained from it is termed "mixed milk." The source of each of these samples is carefully recorded. They are

submitted to microscopical examination and tested by inoculation. The farms supplying milk found capable of producing tuberculosis are inspected; the cows are examined by the veterinary inspector, who takes a sample of milk from each udder that appears to him diseased and possibly affected with tuberculosis. These samples of unmixed milk are tested bacteriologically, and when any is found capable of producing tuberculosis, this is taken as a proof that the corresponding cow is secreting tuberculous milk, and that it is in all probability suffering from tuberculous mastitis. The farmer is required by the medical officer of health to isolate the cow or cows producing tuberculous milk, and at the same time he is advised to have the animal slaughtered in the presence of the veterinary inspector. As this advice is followed in the majority of cases, opportunity is afforded to verify the accuracy of the bacteriological finding, and an important source of infection is removed.

With the sanction of the Health Committee I had from the first certain information relating to each farm and to each cow dealt with entered on forms which I provided for that purpose. Dr. Niven has kindly furnished me with additional data whenever it was possible for him to do so, and with his sanction I have also obtained from Mr. Brittlebank valuable information regarding the state of the inspected cows and cowsheds, and the exact position of the farms which were not clearly indicated on the ordnance map. As a detailed account of my methods and results has recently appeared in the Thirty-Eighth Annual Report of the Local Government Board,¹ it would not serve any useful purpose to burden this communication with similar details. This report—the scope of which was indicated to me by Dr. Newsholme—contains an account of: (1) Experiments which I made between the years 1892 and 1908; (2) methods which I devised before 1895 for administrative purposes; (3) results of the examination, for various sanitary authorities, of 7,000 samples of milk, carried out in my laboratory between the years 1896 and 1908; (4) sources of the tubercle bacilli found in the milk examined; (5) distribution of the farms supplying tuberculous milk. Some details not included in the above report are given in another paper which I read last November before the Manchester Statistical Society.²

For the present purpose it will be sufficient to reproduce here tabulated statements of the results obtained in connexion with the

¹ Supplement containing the Report of the Medical Officer for 1908-9, pp. 341 to 414.

² "The Manchester Milk Supply from a Public Health Point of View," *Transactions of the Manchester Statistical Society*, 1909-10.

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Manchester milk supply only. In the following tables I have brought up to date the returns published in the report and communication mentioned above by the addition of the figures relating to the years 1908 and 1909:—

TABLE I.—SAMPLES OF MIXED MILK COLLECTED IN MANCHESTER DURING THE YEARS 1896 TO 1909 INCLUSIVE.

Years of collection	Total number of samples tested for tubercle bacilli by inoculation		Samples found to be capable of producing tuberculosis by inoculation	
			Actual number	Percentage
Three years, 1896-99 ¹	...	110	19	17.2
1900	...	358	40	11.1
1901	...	440	43	9.7
1902	...	424	38	8.9
1903	...	430	51	11.8
1904	...	425	43	10.1
1905	...	757	60	7.9
1906	...	704	47	6.6
Three years	1907	722	43	5.7
	1908	341	30	8.9
	1909	649	36	5.5
Totals, 1896-1909	...	5360	450	8.3

This table shows that, up to the end of the year 1909, 5,360 cans of mixed milk sent to Manchester were sampled on their arrival, and that 450 of these cans were found to contain tuberculous milk. This makes an average of 8.3 per cent. for the whole period.

TABLE II.—SAMPLES OF UNMIXED MILK OBTAINED BY THE VETERINARY INSPECTOR FROM COWS THE UDDERS OF WHICH APPEARED TO HIM TO BE POSSIBLY AFFECTED WITH TUBERCULOSIS.

These cows were all on farms from which tuberculous milk had been sent to Manchester.

Years of collection	Total number of cows tested		Number of cows affected with tuberculous mastitis	
			Actual number	Percentage
1896-98	...	29	5	17.2
1899	...	19	3	15.7
1900	...	142	31	21.8
1901	...	166	34	20.4
1902	...	95	32	33.6
1903	...	68	28	41.1
1904	...	85	18	21.1
1905	...	108	28	25.9
1906	...	125	33	26.4
1907	...	100	30	30.0
1908	...	74	26	35.1
1909	...	71	22	38.0
Totals, 1896-1909	1082		290	26.8

¹ No mixed samples taken in 1899.

This table shows that up to the end of 1909 the udders of 1,082 cows, among those that had been examined clinically by the veterinary surgeon, showed signs of being possibly affected with tuberculosis, and that of these 1,082 udders 290 were proved to be capable of producing tuberculosis in guinea-pigs by inoculation.

TABLE III.—NUMBER OF FARMS TESTED EACH YEAR, WITH NUMBER OF FARMS SENDING TUBERCULOUS MILK TO MANCHESTER.

	FARMS TESTED FOR THE FIRST TIME			FARMS RETESTED		
	Total number tested	Number found tuberculous		Total number	Number found tuberculous	
		Actual number	Percentage		Actual number	Percentage
1896-1900 ...	427	72	16·8	—	—	—
1901 ...	161	16	9·9	207	28	13·5
1902 ...	131	18	13·8	217	23	10·5
1903 ...	109	12	11·0	230	32	13·9
1904 ...	111	12	10·8	220	26	11·7
1905 ...	209	15	7·1	362	23	9·1
1906 ...	145	9	6·2	410	39	9·5
1907 ...	92	5	5·4	457	35	7·6
1908 ...	60	4	6·6	218	24	11·0
1909 ...	168	13	7·7	446	25	5·6
Totals ...	1613	176	10·9	2767	255	9·2

One thousand six hundred and thirteen farms were therefore among them tested 4,382 times, and were found on 414 occasions to supply tuberculous milk. In the group of farms retested are included farms which were not found tuberculous when first tested, but were found tuberculous afterwards.

Tables I, II, and III are of great interest from an administrative point of view. Table I shows that in the three years 1896 to 1899 (exclusive) 17·2 per cent. of the cans of milk arriving in Manchester contained tuberculous milk capable of infecting experimental animals, and that the average for the three years ending with the year 1909 had been reduced to 6·36 per cent. This result had been in great part obtained by the removal from the herds supplying Manchester of some 290 cows (Table II) proved by bacteriological examination to be suffering from tuberculosis of the udder, and by sanitary measures promoting the health of the cows. The effect of these measures is also indicated in Table III, which shows that the number of farms supplying tuberculous milk is now less than half of what it was in 1900. In order to find out whether this improvement was not attributable to influences other than

the active supervision exercised by the Manchester authority, I selected three quite separate districts, situated respectively in the northern, eastern, and southern parts of the area from which Manchester obtains milk; I then compared the frequency of tuberculous infection of the milk sent from the farms in each of these districts to Manchester, and of the milk sent from neighbouring farms situated in the same districts to other towns. All the samples of milk used for this comparison having been collected according to the same methods, and examined in my laboratory during the same period, the results are strictly comparable. The results are summed up in the following short table :—

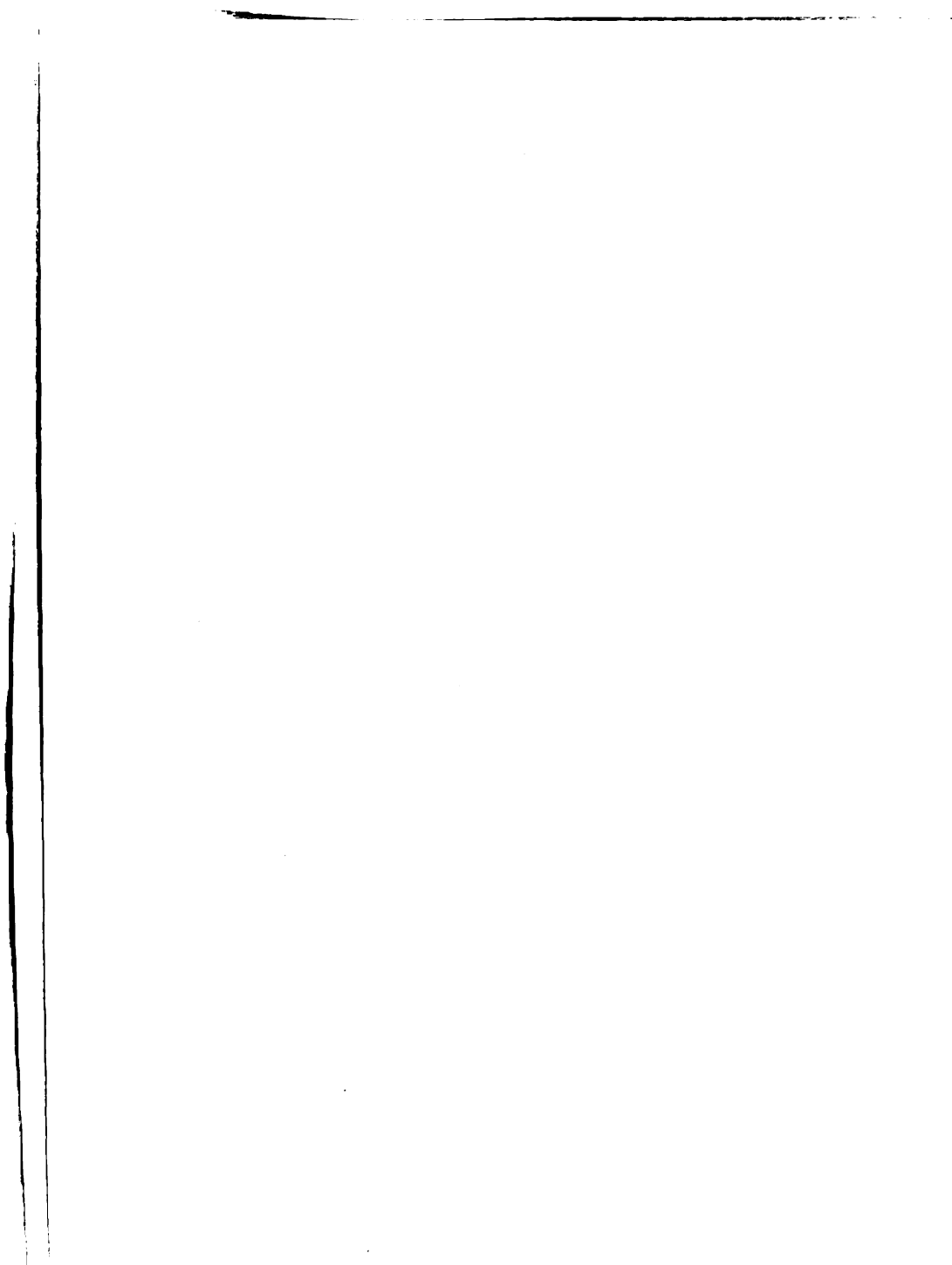
TABLE IV.—COMPARISON OF THE RESULTS OF THE EXAMINATION OF 745 SAMPLES OF MIXED MILK SUPPLIED RESPECTIVELY TO MANCHESTER AND FOUR OTHER TOWNS DURING THE YEARS 1906 AND 1907, AND OBTAINED FROM FARMS SITUATED IN THREE DIFFERENT DISTRICTS.

Districts in which the farms supplying the milk examined are situated	PROPORTION OF TUBERCULOUS MILK SUPPLIED TO EACH TOWN (1906-7).						
	Manchester			Town A,	Town B,	Town C,	Town D,
	111 samples	194 samples	165 samples	49 samples	84 samples	69 samples	73 samples
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Northern district ...	3·6	—	—	4·0	—	—	—
Eastern district ...	—	4·6	—	—	11·9	8·6	—
Southern district ...	—	—	7·2	—	—	—	13·7

It will be noticed that in each case the amount of tuberculous milk reaching Manchester was materially less than that reaching other towns, notwithstanding the fact that originally the milk sent to Manchester from the same districts was infected even to a greater extent than the milk supplied to the four other towns during the years 1906 and 1907. It is also clear that the cows kept respectively in the northern, eastern, and southern districts were very unequally affected with tuberculosis.

The results recorded in Tables I to IV show clearly that the skilful administration of the Manchester milk clauses by our President has brought about a very great improvement in the Manchester milk supply. It is not, however, with this aspect of the question that the present communication is concerned, but with the indications which the figures previously quoted supply regarding the distribution of tuberculosis. They indicate clearly :—

- (1) That various districts are very unequally infected with tuberculosis.



(2) That bovine tuberculosis is preventable to a very material extent.

(3) That at least some of the conditions against which preventive measures have been directed are of importance in relation to the prevalence of bovine tuberculosis.

(4) PROPORTION OF TUBERCULOUS FARMS IN VARIOUS REGIONS.

For the purpose of ascertaining the distribution of tuberculous farms in the region dealt with I have prepared maps in which the position in each of the farms that have been tested between 1896 and 1909 is indicated. I have subdivided these maps, which are based upon the "one-inch-to-the-mile" ordnance map, into one-square-mile areas. These geometrical areas are much more convenient for this kind of investigation than the larger and unequal areas corresponding to administrative districts, the shape of which is also too irregular to allow of accurate comparison being made. The *one-square-mile* areas are grouped in one-mile-broad *longitudinal zones*, numbered consecutively from west to east, and in one-mile-broad *latitudinal zones*, numbered from north to south. The exact position of each one-square-mile area may therefore be accurately determined by two numbers. To facilitate reference I have grouped the longitudinal zones into four-mile-broad zones, each of which is designated by a capital letter. The latitudinal zones are grouped in the same way, and each four-mile-broad latitudinal zone is numbered with a Roman numeral.

The position of each of the tested farms was spotted on this map. The position of the farms which were not indicated by name in the ordnance map has been determined with the assistance of Mr. Brittlebank, whose intimate knowledge of the various districts under consideration has been of very great value to me. Notwithstanding the trouble taken to indicate as accurately as possible the position of the farms not shown in the ordnance map, it was obviously impossible to avoid a certain amount of error; this probably does not exceed quarter of a mile, and in the great majority of cases is much less. In Map I all the farms tested or inspected between 1896 and 1909 are marked; those which have been found tuberculous at one time or another during the thirteen years are indicated by black spots; those that have not once produced tuberculous milk are indicated by rings. The farms in each square are numbered for the purpose of reference.

In summing up the facts brought out by this map I will avoid any reference to the political divisions of the areas under investigation, as

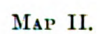
this would serve no useful purpose, and might, as it has done on a previous occasion, give rise to unnecessary annoyance and misconception.

In Table V the situation of the various districts is indicated by the letters and numerals corresponding respectively to the longitudinal and latitudinal four-mile zones. It does not include eighty-nine farms which were tested between June, 1909, and December, 1909. The addition of

TABLE V.—PROPORTION OF FARMS FOUND TUBERCULOUS IN VARIOUS REGIONS.

Latitudinal zones	Longitudinal zones	FARMS TESTED, 1896-1909			TUBERCULOUS FARMS INSPECTED.		
		Number	Found tuberculous	Per cent.	Number	Tuberculous udders found	Per cent.
North of Map							
III	I	3	—	—	—	—	—
IV	M						
IX	L	76	8	10.5	7	4	57.1
X	J, K, L						
XI	D, G, J, K, L						
XII	F, G, I, J, K						
XIII	F	102	15	14.7	12	9	75.0
XIV	F, M						
XV	G, H, J, K, M, N						
XVI	L						
XVII	I, K, L, M						
XVIII	K, L, M, N, O	101	18	17.8	15	13	86.6
XIX	I, J, K, L, M, N, O						
XX	I, J, K, L, M, N, O, P, Q	123	28	22.7	28	16	57.1
XXI	I, J, K, L, M, N, O, P, Q	263	63	23.9	55	43	78.1
XXII	E, F, G, H, I, J, K, L, M, N, P, Q, R	279	83	29.7	80	55	68.7
XXIII	F, G, H, I, J, K, L, M, N, P, Q, R, S						
XXIV	H, I, K, L, M, N, O, P, Q, R, S, T	181	45	24.3	40	26	65.0
XXV	H, I, J, K, M, N, O, P, Q, R, T	143	20	14.9	19	13	68.4
XXVI	H, I, J, K, L, N, O, P, R, S	101	17	16.8	16	9	56.2
XXVII	H, I, K, M, L, O, P	62	14	22.5	14	11	78.5
XXVIII	E, F, G, H, I, J, L, O, P, Q, S						
XXIX	F, G, I, P, Q	90	15	16.6	(12	12	100)
XXX	D, C						
XXXI	Q						
Totals		1524	326	21.3	298	211	70.8

¹ Several of these farms were inspected, primarily, in new districts.



these ninety farms brings the total of inspected farms to 1,613, of which 344, or 21·3 per cent., have at one time or another during the last thirteen years been found to produce tuberculous milk. The addition of the eighty-nine farms does not alter the percentage previously arrived at.

Table V brings out clearly the very unequal distribution of tuberculosis of the udder. To obtain comparable numbers of farms I have, where necessary, grouped together adjacent zones. The effect of this is to reduce the contrast between the best and the worst districts, but, as all the farms in each district have not been tested, this obliteration of excessive contrasts tends to make the figures more reliable. Manchester is included in the latitudinal zones XVII and XVIII; nearly all the farms in that zone are situated either within the town boundary or in the neighbourhood of the town. On nearly 16 per cent. of these farms cows with tuberculous udders have at one time or another been found.

In the sixteen-mile-broad group of Zones IX, X, XI, and XII (North of Manchester) the percentage was	10·5
In the twenty-mile-broad group of Zones XIII, XIV, XV, XVI, and XVII (South of the latter and including the Northern half of the town) the percentage was	14·7
In Zones XVIII and XIX, eight miles broad (including the Southern half of Manchester), the percentage was	17·8
In Zone XX the percentage was	22·7
" XXI	23·9
" XXII	29·7
" XXIII	24·3
" XXIV	14·9
" XXV	16·8
" XXVI	22·5
In Zones XXVII, XXVIII, XXIX, XXX, and XXXI the percentage was	16·6

In attempting to discover the causes which have brought about this unequal distribution it is necessary to consider the possible influence of a number of factors. The area under consideration is not extensive enough to allow of such marked climatic differences as might reasonably be expected to have brought about the unequal distribution of the disease. There is no obvious relation between the distribution of the disease and the geological features of the various districts. I have not, however, analysed yet the facts sufficiently fully to be in a position to express a final opinion upon this point. With regard to the topography of the area there is one coincidence which at once calls for notice. A great part of the land in the region south of Manchester, where tuberculosis was very prevalent, is distinctly at a lower level, on an average, than the northern regions, where tuberculosis was less prevalent. A cursory glance at the map shows, however, that there are several high-level districts that are much affected, and many low-lying districts where farms are comparatively free from infection.

(5) INFLUENCE OF ALTITUDE AND OF VICINITY OF RIVERS.

To study the influence of altitude and other physical features of the land, I have prepared a map of Lancashire and Cheshire (Map II) in which the rivers, the 300 ft. and the 1,000 ft. contour lines are shown. These data are sufficient to indicate broadly the elevation and configuration of the surface of the land in most of the districts in which the farms are situated. The course of the rivers in the lowest regions may be taken to indicate the parts where excessive dampness may be expected. On the same map I have indicated in each square-mile area the number of farms tested, and the number of those which have been found tuberculous at one time or another, between 1896 and 1909. This map shows, therefore, the proportion of farms that have been found tuberculous out of the total number examined in each square-mile area, and at the same time the altitude in each area and its relation to streams.

The information obtainable from this map is summed up in Table VI, according to the same plan as the general results of the inspection of farms have been tabulated in Table V. The number of farms included in Table VI is smaller than in Table V, because Map II takes up only the western slopes of the Pennine Chain in the regions corresponding to Lancashire and Cheshire. This map includes an area containing 1,234 farms out of a total of 1,613 tested up to the end of December, 1909. The percentage of farms found tuberculous in the total area and in the area shown in the map is as follows:—

Total area	1,613 farms, of which 344 were tuberculous, or 21·3 per cent.
Area shown on Map II	1,234	„	271 „ 21·9 „

It is therefore probable that the area shown in the map represents fairly the total area. The advantage of selecting the western slopes of the Pennine range is that all that region is exposed to very similar climatic conditions.

A comparison of Tables V and VI leads one to the conclusion that the greater prevalence of bovine tuberculosis in the districts situated south of Manchester cannot be attributed only to the effects of difference of altitude or of the dampness of the soil attributable to the neighbourhood of rivers. The average percentage of tuberculous farms was:—

On or near the banks of rivers	22·7
On lands at a level not exceeding 300 feet	26·2
On lands at a level of 300 feet	20·8
On lands at a level of 300 to 1,000 feet	19·2

TABLE VI.—PROPORTION OF TUBERCULOUS FARMS IN VARIOUS ZONES AND AT VARIOUS ALTITUDES. THE ZONES ARE GROUPED IN THIS TABLE IN THE SAME WAY AS IN TABLE V.

Latitudinal zones	Longitudinal zones	ALTITUDE OF THE "ONE-SQUARE-MILE" AREAS IN EACH ZONE							
		Squares below the 300 ft. contour line and traversed by rivers	Aggregate No. of farms	Farms tuberculous (percentage)	Squares below the 300 ft. contour line and not traversed by rivers	Aggregate No. of farms	Farms tuberculous (percentage)	Squares situated on the 300 ft. contour line including squares situated above the 1000 ft. line	Squares situated above the 300 ft. contour line. Total including squares situated above the 1000 ft. line (No. of farms transferred to the adjacent column.)
IX - X	L, —, J, K, L	...	12	16.6	10	10.0	19	10.4	31
XI	D, G, J, K, L	...							
XII	F, G, I, J, K	...							9.7
XIII - XIV	F, —, F, M	...							
XV	G, H, J, K, M, N	...	29	10.3	3	(33.3)	44	9.0	24
XVI	L, K, L, M	...							
XVII	I, K, L, M	...							20.8
XVIII	K, L, M, N, O	...	17	6.0	39	23.0	16	18.7	30
XIX	I, J, K, L, M, N, O	...							
XX	I, J, K, L, M, N, O	...	18	33.3	39	30.7	—	—	56
XXI	I, J, K, L, M, N, O	...	2	(50.0)	111	26.1	28	28.9	104
XXII	E, F, G, H, I, J, K, L, M, N	...	11	27.2	101	40.0	47	30.0	86
XXIII	F, G, H, I, J, K, L, M, N	...	11	45.4	11	36.3	17	29.4	72
XXIV	H, I, J, K, M, N, O	...	8	(25.0)	5	(0)	7	(28.5)	51
XXV	H, I, J, K, M, N, O	...	3	(33.3)	23	17.3	4	(25.0)	48
XXVI	H, I, J, K, L, N, O	...	12	(33.3)	11	18.1	8	(33.3)	35
XXVII	H, I, K, L, M, O	...	—	—	7	(42.8)	2	(0)	27
Totals and averages	...	123	22.7	300	26.2	187	20.8	564	19.2
After excluding groups of less than 10 farms	Maxima	—	45.4	—	40.0	—	30.0	—	23.2
	Minima	—	6.0	—	10.0	—	9.0	—	7.4

The differences between the extremes of this series are small compared with the difference between 10·5 per cent. in northern Lancashire and the adjacent Yorkshire districts, and 29·7 per cent. in a region south of Manchester. The farms situated above the 300-ft. line are apparently less liable to tuberculosis than the farms situated below that line, but the difference between 21·3 per cent. and 19·2 per cent., which indicate respectively the average number of tuberculous farms for the whole area and for the farms situated on high grounds, is not very great. A closer examination of the results recorded in Table VI shows still more clearly that the influence of the difference of altitude under consideration is not a dominating factor. If one excludes from consideration the groups consisting of less than ten farms, it will be found that in each altitude there are groups that are comparatively free, while others are extensively affected. The differences between the minima and the maxima relating to each altitude are considerable. Thus, the least affected group of farms (6 per cent.) is found among those situated near the banks of rivers, but in the same category is also found the group showing the highest percentage of tuberculous farms—45·4. The same contrasts are observed in all categories. It is interesting to note that the maxima indicate the same tendency to improvement as the altitude increases, as is shown by the averages. It is, however, quite clear that some more powerful influence than that of situation must be found to explain the considerable differences which I have previously indicated.

(6) INFLUENCE OF THE STATE OF THE COWSHED AND HERDS.

Much has been said of the share taken in the spread of tuberculosis by badly constructed, small, ill ventilated, and otherwise insanitary shippens, of the liability to tuberculosis of certain breeds of cattle, of the pernicious influence of "close breeding for high milking," of the effects of heredity, &c., &c. Although very strong opinions have been expressed by practical and other men upon each of these points, it is difficult, not to say impossible, to discover any satisfactory record of facts in their support.

In the hope of obtaining more definite information, I asked Mr. Brittlebank to give me certain particulars regarding some 400 farms inspected by him. The particulars asked for were the following: State, age, construction, sanitation, cubic capacity, ventilation, cleanliness of cowshed; number of cows kept; how much kept in the open; special breed; state of cows; how long kept on the farm; average age; stability

of stock ; precautions taken to avoid the introduction of diseased animals ; arrangements for dealing with diseased animals and preventing the spread of disease.

I was aware of the difficulty of obtaining definite or accurate answers to many of the questions included in my schedule, but I thought it desirable to ascertain to what extent attention was paid to the various conditions which might affect the health of the cattle. It may at once be said that several of my questions remained unanswered for the simple reason that they referred to matters which, generally speaking, had not received proper attention from the farmer. Thus there is no evidence that, apart from reliance on general appearances and judgment based upon practical experience, farmers as a rule adopt any *reliable method to avoid the introduction of diseased animals among their herds*, or that they make, of their own accord, *systematic provision for the isolation or disposal of tuberculous animals*. It appears also difficult to obtain very reliable information with regard to the *average age or stability of stock* ; this information would have been easily obtained if farmers generally made it a practice to replace animals that had reached a certain age. Very little information capable of forming a reliable basis for generalization could be obtained regarding the *amount of outdoor life* allowed to cattle in various districts. On some farms cattle are kept in the open as much as possible, but I have so little information regarding the majority of farms that it is not possible to estimate how far the practice influences the incidence of tuberculosis. The keeping of cows in the open all the summer and whenever the weather permits is certainly not sufficient to prevent the occurrence of tuberculosis, for I have clear statements regarding ten farms where the practice was adopted, and in seven of these farms tuberculosis of the udder was observed. On two of these tuberculosis of the udder was detected twice. There were, altogether, 125 cows on these seven tuberculous farms, and among these cows eight cases of tuberculous mastitis were clearly demonstrated ; two probable cases escaped detection. Of these seven farms, three were quite satisfactory from a sanitary point of view, one was moderately good, and three were bad.

With regard to the *breed*, there is nothing in the evidence at my disposal indicating that the prevalence of tuberculosis in any district had any relation to the breed of cattle. On the great majority of farms all through the area the herds were composed of animals of various breeds.

In the case of twenty-six out of 292 tuberculous farms there is a clear statement that the whole or part of the cattle were *home bred*. This

is sufficient to prove that the practice is not uncommon, but I have no means of ascertaining whether there was any difference in that respect between tuberculous and non-tuberculous farms. With regard to the *general state of shippens*, their *ventilation* and *cleanliness*, the *general state of the cows*, and their *approximate age*, I have somewhat more complete information. In most cases this information is in the form of an expression of opinion, but, the opinion being that of an experienced veterinary surgeon (Mr. Brittlebank), and generally based on repeated inspection, it is probably more reliable than records of figures collected by less reliable observers. Moreover, it was only after obtaining the whole of the information about farms, not arranged in any definite order, that I classified all the facts, so that no preconceived idea could have materially influenced the record.

Out of 397 schedules relating to an equal number of farms, I had to exclude twelve which were too incomplete to be of any use. This left 385 farms available for the purpose of the investigation. Of the 9,283 cows on these farms, 1,017 have been found to show some evidence or other of abnormal conditions of the udder, and 287 have been proved to be actually suffering from tuberculous mastitis. These results refer to the years 1896 to 1909, during which each of the 397 farms has been inspected more or less continuously during periods ranging from two to twelve years.¹

In estimating the proportion of infected animals it is important to keep in mind the fact that during a period of twelve years the dairy stock of many farms had been entirely renewed. Even in the case of farms which have been inspected during shorter periods, it is probable that the composition of the stock was not exactly the same at the beginning as at the end of the period. This want of stability, and the different practices adopted with regard to the renewal of old stock, make it impossible to estimate exactly the case incidence of tuberculous mastitis. The estimate of the proportion of infected farms is not vitiated to the same extent by the movements of the stock, although it is obvious that the chances of infection by the occasional introduction of tuberculous animals are

¹ The periods during which the farms were inspected are as follows:—

One to two years	153 farms
Three to four years	81 "
Five to six years	49 "
Seven to eight years	56 "
Nine to ten years	41 "
Eleven to twelve years	17 "
					<hr/>
					397

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increased when animals are frequently bought and sold. On the other hand, it is well to remember that when cows are allowed to age on an infected farm, the proportion of tuberculous animals increases with the age of the stock.

I have published elsewhere statistics relating to several herds, in which I found that less than 50 per cent. of the cows under five years were affected with tuberculosis, while more than 50 per cent. of the cows above five years of age were affected with tuberculosis. These statistics were based upon the results of the testing of 362 cows with tuberculin, confirmed by post-mortem examination in every case.¹ Whatever may be the disturbing influences of these various irregularities, it is obvious that if the situation of the farm, the state of the shippon, and the general condition of the animals, have a material influence upon the prevalence of tuberculosis, this should appear clearly when the characters of the farms which have been found free from tuberculosis are compared with those of the tuberculous farms.

To facilitate this comparison, I have tabulated the information at my disposal as follows:—I have divided all the farms into two groups: (a) Farms which were never found to produce tuberculous milk, (b) farms which were found to produce tuberculous milk. The farms (77) included in Group A, not being numerous enough for subdivision into many latitudinal groups, have been divided into two divisions, the first corresponding to latitudinal Zones III to XIX, and including the Manchester farms with the exception of the suburban farms on the south of the city. The second division corresponds to latitudinal Zones XX to XXXI, and includes all the farms situated south of Manchester. The number of farms (385) included in Group B was large enough to be subdivided into five divisions. The first division of this group (III to XIX) corresponds exactly to the first division of Group A, and includes all the Manchester farms and those situated north of Manchester. It also corresponds to the three upper divisions in Table VI. The other four divisions correspond to the nine lower divisions in Table VI, and comprise all the farms south of Manchester, including the southern suburban farms. The data relating to the farms situated in each of the divisions are classified as follows:—

(I) *State of the cowshed* as indicated by (1) general condition of the buildings; (2) cubic space and ventilation under the head of ventilation; (3) cleanliness.

¹ Report of the Medical Officer to the Local Government Board, 1908-9, pp. 408-9.

(II) *State of the cows*: (1) approximate age; (2) general health and nutrition, generally spoken of as condition of the animals.

(III) *Number of cows* on the farm.

(IV) *Number of cows showing some sign of disease of the udder*.

(V) *Number of cows proved to be affected with tuberculous mastitis*.

In order to indicate the state of the shippoon and of the stock, I adopted the following device. The state of things recorded under each question in the schedules was classified under three heads: (1) Very good, good, or satisfactory; (2) fair, moderate, or indifferent; (3) very bad, bad, unsatisfactory or poor. This classification was not practicable with regard to the age, which I grouped as follows: Young or under 5; uncertain or middle-aged, or mixed when the proportion was not given; old, or over 8 years. Old does not, of course, mean that all the cows kept in the shippoon were old, but that many were.

Having thus ascertained for each category the proportion of shippoons that were unsatisfactory or bad beyond doubt, I found the proportion existing between these unsatisfactory shippoons and the total number of shippoons available for the study of each special feature. The shippoons with regard to which I had no clear information, either good or bad, were excluded. The results of this part of the investigation are summarized in Table VII (pp. 236-7).

Before proceeding to a discussion of the meaning of the facts recorded in Table VII, I must explain that the shippoons included in the latitudinal Zones III to XIX in Groups *A* and *B* are nearly all situated in Manchester, or in the immediate neighbourhood of the town, and that they are those remaining after a great number of unsatisfactory shippoons had been closed during the first years of the period under consideration. These farms are, therefore, selected farms; moreover, most of them have been improved under the supervision of the Health Department.

With the exception of some farms in Zone XX, very few of the other farms included in the table were so completely under the control of the authority. The contrast between the two classes of farms is well shown by the two Divisions III to XIX and XX to XXXI, in Group *A* (non-tuberculous farms); the contrast is also evident when Division III to XIX is compared with the four Divisions XX to XXXI in group *B* (tuberculous farms). The averages for *A* and *B* are distinctly affected by the improved state of the Manchester farms. An idea of their original state may, however, be gathered from the figures showing the number of cows, the udders of which were under suspicion when the farms were first examined.

TABLE VII.—RELATIONS BETWEEN THE STATE OF THE SHIPPON, THE CONDITION

Farms grouped according to the occurrence or non-occurrence of tuberculous infection of the milk between 1896 and 1909	Latitudinal zones	(I) STATE OF THE COWSHEDS								
		(1) General condition of the buildings			(2) Ventilation			(3) Cleanliness		
		No. of cowsheds available	No. of cowsheds bad	Percentage	No. of cowsheds available	No. of cowsheds bad	Percentage	No. of cowsheds available	No. of cowsheds bad	Percentage
(A) Farms which were never found to produce tuberculous milk	III - XIX	38	3	7	38	2	5	38	1	2
	XX - XXXI	35	24	68	34	23	67	35	20	57
	Whole area,) III - XXXI)	73	27	37	72	25	34	73	21	28
(B) Farms which were found on one or more occasions to produce tuberculous milk	III - XIX	39	17	43	33	16	48	33	10	30
	XX - XXI	75	34	45	75	43	57	75	28	37
	XXII	71	37	52	69	37	53	70	38	54
	XXIII - XXIV	58	47	81	58	46	79	58	43	74
	XXV - XXXI	41	26	63	40	29	72	41	28	68
	Whole area,) III - XXXI)	284	161	56	275	171	62	277	147	53
Totals for all the farms	357	—	—	347	—	—	350	—	—
(C) Farms belonging to various districts and which were found to produce tuberculous milk on more than one occasion. (These farms are also included amongst the 284 farms of Group B)	III - XXXI	49	26	53	48	28	59	49	17	34

AND NUMBER OF COWS, AND THE PREVALENCE OF TUBERCULOUS MASTITIS.

(II) STATE OF THE COWS						(III) No. OF COWS ON FARM		(IV) No. OF COWS SHOWING SOME SIGN OF DISEASE OF THE UDDER		(V) No OF COWS FOUND AFFECTED WITH TUBER- CULOUS MASTITIS		No. of farms in each group
(1) Approximate age			(2) General health and nutrition									
No. of cowsheds available	No. in which old cows numerous	Percentage	No. of cowsheds available	No. in which state of cows bad	Percentage	Total No. of cows in each group	Average No. of cows per farm	Actual No.	Percentage	Actual No.	Percentage	
37	1	2	38	0	0	595	15	43	7.2	—	—	38
29	17	58	35	10	28	972	24	45	4.6	—	—	39
66	18	27	73	10	13	1567	20	88	5.6	—	—	77
23	6	26	29	4	13	814	20	102	12	33	4	39
56	32	57	69	11	16	1848	22	266	14	84	4	82
57	27	47	67	13	19	2133	26	249	11	66	3	80
41	27	65	55	16	29	1661	25	192	11	57	3.4	64
31	20	64	37	12	32	1260	29	130	10	47	3.8	43
208	112	53	257	56	21	7716	25	989	12	287	3.7	308
274	—	—	330	—	—	9283	—	1017	—	287	—	385
39	23	59	43	9	21	1164	23	259	22	75	6	—

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Some of the facts brought out by Table VII may be summarized as follows:—

Average state of farms and stock as indicated by the number of unsatisfactory shippens per cent. of the total available for comparison		
	Shippens producing non-tuberculous milk	Shippens producing tuberculous milk
Bad general condition of the building ...	37 per cent. ...	56 per cent.
Size and ventilation insufficient ...	34 ...	62
Want of cleanliness ...	28 ...	53
Many cows old ...	27 ...	53
Bad condition of stock ...	13 ...	21
Average number of cows with suspicious udders ...	5·6 ...	12
Average number of cows with tuberculous udders ...	0 ...	3·7
Average number of cows per farm ...	20	25

It would appear from this that in every respect the shippens which were found to produce tuberculous milk were worse than the others. But if, instead of comparing the averages only, one compares in each group the state of the tuberculous and non-tuberculous farms in various latitudinal zones, it becomes evident that the averages do not convey the whole truth, for if the non-tuberculous farms situated south of Manchester (Group A, Division XX to XXXI) are compared with the tuberculous farms also situated south of Manchester, one finds that the tuberculous farms in Zone III to XIX, XX to XXI and XXII are really more satisfactory than the non-tuberculous farms in Zone XX to XXXI, and that it is only south of Zone XXII that the tuberculous farms are worse in every respect than the non-tuberculous farms (not including the Manchester farms).

Another unexpected fact is obvious. Of the five divisions of tuberculous farms, the one corresponding to Zones XXIII and XXIV is undoubtedly the worst, while Division III to XIX is distinctly the best; but notwithstanding the considerable differences indicated in the table, the number of cows with tuberculous udders per cent. of the total number of cows is about the same in all the zones. The same may be said of the cows suspected of disease of the udder: there are slight differences, but these do not correspond in the least with the condition of the farms in the respective zones. I do not think that the differences in these percentages should be taken to indicate important differences in the incidence of tuberculosis, as they may possibly be affected by the more frequent inspection of the proximal farms, which would almost certainly lead to the discovery of some few cases overlooked in the first visits.

It is, however, clear that the condition of the farm, though important, is not the dominant factor, and this makes it necessary to believe in the existence of some cause capable of overpowering the most favourable conditions, and practically independent of the most influential predisposing factors, to account for the focal distribution of cases of tuberculous mastitis.

(7) ACTIVELY AND POTENTIALLY INFECTIVE CASES OF TUBERCULOSIS.

A careful consideration of the statistical facts recorded in this paper, and of facts which I have observed in connexion with a number of farms that I have been able to study closely, has led me to the conclusion that cows in a state of advanced tuberculosis, and *emitting discharges loaded with tubercle bacilli*, constitute the chief factor determining the distribution of bovine tuberculosis. The number of cows suffering from tuberculosis is so great that there are very few farms that are entirely free from tuberculosis, but so long as the tuberculous lesions are confined to the serous membranes, bones, lymphatic glands, or viscera, and have not ulcerated, *they retain their bacilli*. Cows affected with these closed lesions, though potentially infective, are not important sources of infection. When, however, the disease has given rise to ulcerative lesions of the lungs, alimentary passages, genito-urinary passages, or udder, products containing tubercle bacilli are discharged by the animal, the surroundings of which become rapidly infected. As the disease advances there comes a stage when the quantity of tubercle bacilli so discharged is inconceivable. To obtain some information upon this point I have carefully watched a cow affected with pulmonary tuberculosis. For more than six months before its death it had a troublesome cough and at times expectorated some muco-purulent discharge; this was not, however, a conspicuous feature of the case. For four months the milk obtained from two quarters of its udder was loaded with tubercle bacilli, and during the first two out of these four months the supply was abundant. After a time a third quarter began to secrete tuberculous milk. One month before it was killed this cow begun to suffer from incoercible diarrhoea, which continued till its death. The quantity of fluid faecal discharge evacuated by this cow was considerable, and yet almost every drop of this fluid was loaded with tubercle bacilli. The whole floor of the shippon was either covered or splashed over with this fluid, and if I had not prevented it, the litter soaked with this tuberculous material would have been thrown upon the adjacent pasture land on

which other cows were grazing. I have not measured exactly the amount of fluid fæces evacuated by this cow, but estimated roughly that the amount exceeded one gallon a day. It is easy to conceive how the presence of such a cow in a herd even for a period of a few weeks would be sufficient to infect both shippon and pastures to an extraordinary extent. In a well-kept farm such a cow would probably be isolated fairly early, but not before some mischief had been done. In a spacious, well-ventilated, clean shippon, tuberculous discharges would probably not be allowed to accumulate and to contaminate to a very material extent the food or litter of the other cows. In a badly constructed, ill-ventilated, dirty shippon, infection would be practically certain. But whether the shippon was clean or dirty, it is probable that no precautions would be taken with regard to the litter and washings from the shippon, and that considerable quantities of manure would be infected by admixture with this material. Apart from this, the cow might infect pastures directly, and this infection would persist for a considerable time. It is generally known that desiccation or putrefaction do not affect the vitality or virulence of the tubercle bacillus for weeks and months. I have made an experiment which proves that sero-purulent discharge from a tuberculous udder *kept for nineteen months* (part of the time at the ordinary external temperature, but mostly at temperatures varying between -4°C. and $+6^{\circ}\text{C.}$) was still virulent at the end of that period. This milk had been left in the bottle in which it had been originally collected, and it contained other bacteria than the tubercle bacillus. The tubercle bacilli had retained their usual characters and reactions, and their virulence was not very materially diminished. They gave rise to very extensive lesions in inoculated animals.

The extraordinary masses of bacilli which can be emitted by a cow in a state of actively infective tuberculosis, and the great resistance of the tubercle bacilli, whether they be contained in dry or moist products, are, I think, sufficient to explain the comparative independence of tuberculous infection from predisposing causes. I say comparative, because I do not wish it to be inferred that predisposing causes are without effect: I have given evidence to the contrary. I was so convinced in 1897 of the effects of infection of shippons and pastures by cases of advanced tuberculosis that in conducting the Ford Bank experiment¹ I began by eliminating all cases of advanced tuberculosis, separating from reacting cows all the animals that did not react with tuberculin, housing them

¹ "The Stamping-out of Bovine Tuberculosis," *Trans. of the British Congress on Tuberculosis* (State Sect.), Lond., 1902, ii, pp. 235-82.

in sheds that had been thoroughly disinfected, and not allowing these animals to graze in pastures that had already been used by tuberculous animals. New stock that had not yet passed the tuberculin test twice was not allowed to mix with these sound cows. In this way I cleared from tuberculosis a herd of twenty-seven head of cattle in one year, and no fresh case occurred during a second year, although over 61 per cent. of the original stock were tuberculous. The same method has been used since under the supervision of Mr. Brittlebank at a large farm (120 cows) supplying milk to the hospitals under the Manchester Corporation. This farm has now been kept free from tuberculosis for several years.

I am convinced that the success of the work done by the Manchester Sanitary Committee for reducing the amount of tuberculous milk supplied to the town is mostly due to the removal of actively infective cows and to a diffusion of the knowledge of the danger connected with the keeping old cows.

(8) GENERAL CONCLUSIONS.

The evidence analysed in the previous pages, though incomplete, is, I believe, the best available at the present time for the purpose of estimating the causes of the unequal distribution of bovine tuberculosis. Notwithstanding the incompleteness of my data, I believe that certain conclusions can be based on them, and I think that anyone willing to take the trouble of considering fully the facts collected in this communication will find it difficult to come to other conclusions without neglecting some of the facts. If I am mistaken, there shall still remain to me the consoling thought that I have brought before you a number of facts that cannot fail to prove useful to others interested in the prevention of tuberculosis.

There might be some justification for my taking this opportunity to insist once more upon the advisability of adopting the thorough measures which I have advocated during the last fourteen years for stamping out bovine tuberculosis. I think, however, that it is undesirable to lengthen this communication.

The evidence obtained so far shows that bovine tuberculosis is, on an average, more prevalent in districts where shippens are generally in a bad state, small or badly ventilated and dirty, and where also it is the usual practice to retain many aged cows on the farms. It is, however, equally clear that there are districts where the farms exhibit all the

defects above mentioned, and yet have remained free from tuberculous mastitis; on the other hand, there are districts in which the farms were free from these defects, and where many cases of tuberculous mastitis have been observed.

It does not therefore appear safe, under the present conditions, to rely chiefly upon ordinary sanitary measures for the purpose of controlling bovine tuberculosis. The partial or complete failure of ordinary sanitary measures indicates that the action of the infective material is more or less independent of these measures, when it is either very abundant or very virulent. There was no reason to believe that the virulence of the tubercle bacillus was materially affected by the localities investigated, but we know that cows with ulcerating lesions—i.e., cows in the *actively infective stage* of tuberculosis—are capable of emitting, and do emit, an extraordinary number of tubercle bacilli.

From this I am led to the conclusion that *bovine animals suffering from ulcerative tuberculous lesions, more specially of the respiratory organs, alimentary canal, genito-urinary organs and udder, constitute the chief factor determining the excessive prevalence of bovine tuberculosis in certain districts.*

This conclusion indicates the great importance of *inspecting every animal*, of removing without delay all those that are actively infective, and of not allowing healthy animals to remain in places that have been infected, so long as these places have not been treated so as to remove as far as practicable the dangers of infection. All animals affected with tuberculosis are potentially dangerous, and as their age advances the chances of their becoming actively dangerous increases; it is therefore desirable, so long as bovine tuberculosis has not been stamped out, to reduce as much as possible the number of old animals.

I hardly need to attract your attention to the bearings which these conclusions have upon the control of human tuberculosis. The importance of the removal of infective cases of tuberculosis indicates the desirability of segregating cases of advanced tuberculosis.¹ These cases generally are actively infective, and I am inclined to believe that more could be done towards the future control of human tuberculosis by providing suitable homes for infective cases than by the treatment of

¹ "It is obvious that segregation is necessary only when patients are unable, owing to their circumstances or education, to observe or to understand the precautions which they must take to protect their neighbours. I need not say that I am not an advocate of harsh measures of segregation. I have previously advanced the same views in an address delivered before the North Western Branch of the Society of Medical Officers of Health."—*Public Health*, April, 1899.

early cases. Both things are good, but prevention would ultimately render treatment unnecessary and prevent an amount of suffering that treatment can only reduce. The provision of means of prevention need not interfere with the provision of means of treatment.

ADDENDUM.

To prevent any misconception as to the bearing of this communication upon the question of the administrative prevention of bovine tuberculosis, I wish to state emphatically here that in my opinion none of the facts brought out in this paper would justify the view that the adoption of partial measures can be trusted to yield satisfactory results. To make my position clear I repeat here some of the conclusions given at p. 414 of the Annual Report of the Medical Officer to the Local Government Board for 1908-9 :—

(18) So long as the presence of tuberculous cows, and more especially of aged tuberculous cows, is tolerated in our herds, a certain amount of tuberculous infection of the milk supply is inevitable. The elimination of cows with tuberculous udders undoubtedly removes the most material and dangerous source of infection, *but it is only after the milk has become infectious* that these cows are detected. *Frequent inspection* is therefore indicated under the present system of control.

(19) Preventive methods based upon the state of the milk or of the udder cannot give results equal in value, either from an agricultural or from a public-health point of view, to those that could be obtained by methods having for object the *eradication of bovine tuberculosis*. The latter, though more costly at first, would yield more permanent benefits, and finally be less onerous.¹

(20) Measures having for object the control of milk supplies, to be efficient, must be carried out uninterruptedly year after year, very systematically, and over fairly *extensive continuous areas*.

¹ As this aspect of the question is not discussed in the report, I must refer the reader to a paper bearing upon it in the *Transactions of the British Congress on Tuberculosis* (State Section), Lond., 1902, ii, pp. 235-282.

DISCUSSION.

The PRESIDENT (Dr. Niven) said the Section was extremely indebted to Professor Delépine for his elaborate and careful analysis of such a great mass of material. He must have expended very great labour in going through the details and sorting them out. The careful observation of the facts day by day for thirteen years, and the arrangement of them so as to bring out the chief points, constituted a very fine piece of work.

Dr. C. J. MARTIN, F.R.S., in response to the President's invitation, said he was scarcely prepared to open the discussion. It was true that, indirectly, he had had some experience of the examination of tuberculous milk during the last sixteen or eighteen months—namely, milks imported into London which had been forwarded to the Lister Institute by the London County Council. If he had known he would have been called upon to speak, he would have brought some figures. However, he could give approximately the general experience of the examinations. The number of samples of London milk examined had been nothing like the number examined at Manchester by Professor Delépine, but they totalled about 3,500 samples. The test consisted of the usual inoculation of guinea-pigs. The milks were all mixed ones, taken from churns; and the experience of the gentleman who had that matter in charge was that microscopical examination afforded no information of value with regard to whether the milk contained tubercle bacilli or not. He believed that only in about 8 per cent. of the milks which were found, by inoculation, to contain tubercle bacilli, the bacilli had been found by the microscope. The proportion of milks containing tubercle bacilli fluctuated considerably in the markets which supplied London. In successive series of 500 samples it hovered about 10 per cent. He feared that, notwithstanding the energetic action of the Council's officers, the results had not so far shown any material improvement during the nineteen months. That summed up all the special knowledge he had on the subject; everything else was what he had learned from friends who had had greater opportunities and experience concerning milk; and amongst those he included Professor Delépine, to whom a great debt was due for having during the last ten years accumulated such valuable data on the subject. Professor Delépine was peculiarly fortunate in having been associated with such a progressive city as Manchester, and such an enlightened health authority. Professor Delépine's principal thesis that evening, although he had traversed a much wider range, was the distribution of tuberculosis in farms in the neighbourhood of Manchester; and he had endeavoured to analyse what data he had at his disposal as to the correlation between the proportion of tuberculous farms and any other facts. It seemed unfortunate for that purpose, although it was inevitable in the nature of the data, that a red spot meant a farm which was found to have tuberculous cattle over a period of ten years. Many of those farms, he presumed, must have been

examined on more than one occasion. So that there was a collection of farms some of which might have been examined many times, and others which might have been examined only once. So there would be a tendency to find tuberculosis in the neighbourhood of Manchester and the other places which were easily accessible for frequent examination. He gathered that Professor Delépine's opinion was that there was a definite correlation between tuberculosis in the farms and general mismanagement of those farms. One point which had particularly interested him was the apparent correlation indicated between tuberculosis and the age of the cows kept; there seemed to be a tendency in the less well managed and presumably less flourishing dairies to keep old cattle. Tuberculosis in cattle differed from the disease in man in that it was usually not acute, but progressed gradually, and was at its worse in old age. He was ignorant as to precisely how long, in health, a cow could be used economically for dairy purposes, but he believed it to be not more than six or eight years.

The great obstacle to the eradication of tuberculosis which had to be contended with was that a young tuberculous cow which reacted to tuberculin had a fairly long economic career in front of it, if one neglected the risk of having tubercle bacilli in the milk. He would be glad if Professor Delépine or anyone present would tell him of further reliable data as to the proportion of dairy cattle which were the subjects of tuberculosis—i.e. reacted to tuberculin.

Sir SHIRLEY MURPHY said there was such a wealth of detail in the paper that it was difficult to discuss the whole of it. Professor Delépine was to be congratulated on the enormous amount of labour which he had put into the contribution, and the thesis would stand as a work of reference for any other community which desired to work on the same lines. The most hopeful feature about the figures presented was the reduction, not only in the proportion of farms which had tuberculous milk, but in the lesser degree of infection of the inoculated animals as manifested in the later years. If one could translate that into less risk of infection of human beings, it should be a considerable factor in improved health among the people of Manchester. He feared that London could not be dealt with on precisely the same lines of record; the Metropolis drew its milk from such a wide area that it would take a long time to accumulate anything like the facts which Professor Delépine had placed at the disposal of the profession concerning Manchester. He wished Professor Martin had had an opportunity of looking over his notes, showing the results of his examinations of tuberculous milks; but perhaps the Section could hope for something from Professor Martin at a later date.

Mr. M. GREENWOOD, Junr., said he would like to mention one or two minor statistical points. One was as to whether there was any relation between altitude and the prevalence of tuberculosis. Professor Delépine had stated that that relation was a very small one, but he (the speaker) believed a

definite relation had been established by Professor Delépine's work. For example, if one took Table VI and reduced the figures from the percentages, and instead of making a division into three or four groups one divided them into two, which was a statistical advantage, because the two groups had comparable numbers in them, both of which were large, it worked out as follows: If one took the farms at the 300 contour line or over, there were 751 of them, of which 147 showed tubercle, while below the 300 line there were 483, of which 122 were tuberculous. Expressing these figures as percentages, the lower-level farms showed 25·26 per cent. and the higher-level farms 19·57 per cent. of disease. Of course, the difference was not very large, but it was more than a quarter of the smaller value, and the actual number of observations was fairly large, so that the difference found could scarcely be regarded as within the statistical error.¹ Working it out more elaborately and determining the correlation between altitude and freedom from tuberculosis, this came to about 0·13, which was extremely small, but yet nearly three times the size of its probable error, and suggested there was a slight positive relation between the two events. The fact that large numbers of cases occurred in high altitudes was not unfavourable to the conclusion, but simply suggested that in a given area, other things being equal, altitude was favourable. With regard to the last table, he was wondering whether the influence, which, he gathered from Professor Delépine, was small, between the condition of ventilation and cleanliness, &c., and tuberculosis would not be better shown by a sort of intensive study of the figures, rather than by those general tables, because they brought out the enormous differences when taking facts from different areas. For example, simply taking the last column and considering the farms upon which tuberculous udders had been found, it suggested that the dirtier the farm, the smaller the percentage of tubercle existing in it, because the first two gave 4 per cent. of tuberculous mastitis, the corresponding percentages of dirty farms being 30 and 37, while the groups with 54, 74, and 68 per cent. of dirty farms gave 3, 3·4, and 3·8 per cent. of tuberculous mastitis. He would have thought that the data from adjacent squares might be worked out, for example, 22, 23 and 24; there were in them 129 cowsheds and a considerable number of cattle. From the information collected on Professor Delépine's schedules, one could get a measure of the relation between the various conditions, the general management of the farms, and the prevalence of tuberculosis within the area in adjacent squares—i.e., the heterogeneity would be eliminated. In that area it might be possible to measure by the method of correlation the relation between dirt and the progress of the disease. In the general figures, however, that connexion, if it existed, was quite obscured.

Dr. MEREDITH RICHARDS asked whether Professor Delépine could say what results one might hope to obtain if the clinical method were adopted of eliminating tuberculous cows from herds—i.e., if the plan were adopted of getting a quarterly inspection of dairy cows by a veterinary surgeon. He meant not as

¹ 5·7 ± 1·3.

regards elimination of tubercle from the whole of the stock, but what the immediate effect would be on the milk supply. For some years past they had tried in their own sewage farm to get a tubercle-free herd, but so far the results of that had been disappointing, for it had happened from time to time, when some of the cows were slaughtered, that they had been in an advanced stage of tuberculosis. He did not know how that was to be accounted for, unless it was that they ate sewage-contaminated grass.

Dr. G. S. BUCHANAN desired to add his tribute of admiration to that expressed by other speakers concerning the paper. Looking to all the enormous work represented by the paper and the interesting general results which had been set forth, he could not help the reflection that if Professor Delépine had had at his disposal statistics of information which had been collected systematically from farms in particular areas, all of which had been examined by the same method and could be compared, the results would have been even more valuable. He wished to ask a question concerning the evidence of there being something like a definite factor of infection belonging to particular districts or neighbourhoods. He gathered that Professor Delépine considered there were different degrees of infection characteristic of different areas; for example, there was a great difference in regard to prevalence of udder tuberculosis between the districts north of Manchester and those south of the city. If a particular farm adjoined a farm which had infection, was that particular farm assumed to be more likely to give infected milk than a similar farm in another neighbourhood, and surrounded by farms which were free from tuberculosis? That seemed to be important in connexion with certain of the arguments used in the paper. The square areas were judged, and conclusions were drawn, from farms which happened to send to Manchester milk which was detected to be tuberculous. Might it not be a very fortuitous matter in groups of such squares what proportion of those farms sent infected milk to Manchester? Again, there was some difficulty in drawing inferences from the facts in Manchester and those in other towns, such as Derby. A district which sent milk to Manchester was compared with one which sent milk to Derby by the results of the examination of the milk in those places, but it did not appear that the farms in question were the same, or comparable, and one did not know one was comparing anything like the same proportions of milk. With difficulties of that sort, which were inseparable from such an investigation on the data at present available, it was going somewhat too far to enter into speculations from the figures, as Mr. Major Greenwood did, concerning the effect of altitude, &c. If, however, the paper possessed speculative features, it would be admitted that there was no one whose work gave him a better title to indulge in them than Professor Delépine, and that his impressions on these matters must be of great utility.

Professor SIMPSON asked whether the statistical matter was followed up by a history of the cows in the several farms in regard to their origin and movements, and in relation to other infected cows. He wished to add his

congratulations to the author. If such a map as he had published with the paper were put in the House of Commons, he thought the members of that House would be anxious to pass the Milk Bill more readily than they seemed disposed to do.

Dr. COUTTS said he had been associated with Professor Delépine in the early days of this investigation, and what struck him on hearing the paper was the great perspicacity which he had shown in his methods; at the beginning he adopted the methods which he had carried through to the end. He must have surveyed the question very carefully before commencing his investigations, and the fact that there had been no change of method made his statistics of all the more value. He wished to ask the author whether he had been able to differentiate, or trace how far any error in the examination of the mixed milks had occurred through infection of mixed milk through tuberculous material from the bowel, as apart from infection from the udder. At times mixed milk had been found to be tuberculous, and on going to the farm the inspector had failed to find a cow with a tuberculous udder.

The PRESIDENT repeated the indebtedness which the Section was under to Professor Delépine for his mass of careful work. Whatever the defects in the material upon which the Professor was working, and unfortunately much of that material was not as certain as the Professor's own experiments, he was fairly entitled to say that he had established the ætiological point that infection was a dominant element. A word used by Mr. Greenwood, Junr., might profitably be extended in the matter—namely, "management," and it might be held to include the feeding of the cows. There seemed little doubt that the manner in which a farmer fed his cows largely influenced the amount of tuberculosis which prevailed. The age of the cows was also important. Old cows were much more liable to tuberculosis than young ones, and a farmer who knew his business tended to collect young cows; so that if in the Manchester district there were a lowering of the average age of the cows, there would be less liability for tuberculous milk to be imported into the city. That was what had largely happened, owing to the work done by veterinary surgeons in advising farmers to reduce the age of their cows. And that received additional weight in that Professor Delépine had shown, in his experiments with a herd, that in that way alone could the operation of the eradication of tuberculosis from herds be economically conducted. So it was not merely that the farmer ceased to supply tuberculous milk to the same extent as formerly, but in a more economical manner, by lowering the age of the cows. The third important matter was the cleanliness of the building and the enforcement of that cleanliness: that was of vital moment. If the analysis brought one round to that point, it was in consonance with commonsense. It was of great value to have had such a critical survey of the subject.

Dr. S. DELÉPINE: In replying to the remarks which have been made, I must, in the first instance, thank the President and the various speakers for the kindness and generosity with which they have treated my communication. For the purpose of indicating the source of my data I have been obliged to refer to many points which could not possibly be discussed in the time at my disposal, and several of these side issues have been made the subject of remarks which will force me to include in my answers subjects which did not form an essential part of my paper. The object I had in view was to discover whether the data which I had accumulated in connexion with the examination of cows' milk gave some indications as to the factor or factors which had the greatest influence in determining the remarkable inequality of distribution of bovine tuberculosis, tuberculous mastitis being taken as the index. In preparing this paper my aim was to avoid the administrative aspect of my work, and to discuss bovine tuberculosis from a purely epidemiological point of view. It seemed to me that the outcome of such an investigation might not be without bearing upon the question of human tuberculosis. I could not, however, avoid referring to the administrative work done in Manchester, as it was obviously necessary that I should clearly indicate the source of my data. I was much interested by Dr. Martin's remarks; his experience with regard to the testing of samples of milk sent to the Lister Institute by the London County Council is very similar to the Manchester experience between the years 1900 and 1904, so far as the percentage of tuberculous milk is concerned. The statistical results obtained by comparing successive series of 500 samples of mixed milk cannot be expected to show any regular tendency so long as the whole, or the greater part, of the London milk supply has not come under control. Sir Shirley Murphy has to deal with such an immense area that it will take him a considerable time to cover the whole field. He can be heartily congratulated on the very large amount of work which has been done during the last two years. The number of samples tested yearly in London is at present about four times the number examined in Manchester each year, and yet this number is proportionately to the population smaller than that examined in Manchester. Before the results obtained in London can be fairly compared with those obtained in Manchester, the number of samples examined in London will have to be considerably increased.

It is much to be regretted that Parliament should have withheld from Sir Shirley Murphy the additional weapons which would have given him the means of making better use of all this work than can be done under the present conditions. In this Manchester is certainly more fortunate. The magnitude of the task of supervising the milk supply of large cities is such that one is driven to the conclusion that much of the work of direct inspection must ultimately be conducted by authorities having a direct control over the milk-producing districts; this would leave much for the municipalities to do, for they would still have to satisfy themselves that the local work was well done, and if the inspection of cowsheds by county and other local authorities were conducted

by experts, trusting to mere clinical inspection, and not able to use the more accurate methods of diagnosis, this system would give so little guarantee of security that the work of municipalities would not be materially reduced.

I fully agree with Dr. Martin when he says that it was unfortunate, though inevitable from the nature of the data, that the spot map of tuberculous farms should relate to a period of over ten years. Obviously it would have been much easier to deal with the results if the 30,000-odd cows housed in some 1,613 farms had all been inspected in one or two years, and if all the milk had been tested during the same period. It would then have been possible to get a very exact idea of the distribution of tuberculosis *at a given moment*. But unless all the cows had been tested with tuberculin, and the old cows submitted also to a complete clinical examination, the majority of cases of tuberculosis would have escaped detection. The examination of milk can only be used as the means of finding a certain proportion of cases of more or less advanced tuberculosis, and, as I have pointed out in my paper, the only accurate administrative method available in England at the present time for the purpose of estimating approximately the prevalence of tuberculosis is the bacteriological examination of the milk. If all the farms had been examined in a short period, many tuberculous cows, whose udders had not yet, at the time of inspection, become diseased, would have remained undetected. I think, therefore, that the disadvantages attached to the longer period are more than compensated by the advantages derived from the repeated examination of farms. The fact that some of the farms were inspected more often than others does not affect the results as much as would appear at first sight. I naturally paid much attention to this point, and have prepared tables and maps corresponding to several short periods into which I have subdivided the whole period of thirteen years. The indications given by these fractional statistics are the same as those given by the lumped figures for the whole period. This is true of every part of the milk-supplying areas, and is well shown in the reproduction on a larger scale of one of the 16-square-mile areas, entering into the composition of the map of the whole area¹ (see diagram, p. 251). The inspection of the farms during the whole period has been so managed that most farms have been inspected several times at intervals sufficient in most cases to permit of the extension of tuberculosis to the udder in the case of cows that were only slightly affected with tuberculosis at the time of the earlier visits. It is not easy to differentiate clearly in the general map the farms which have been found tuberculous only once and those that have been found tuberculous more than once; this, however, is not necessary to indicate the distribution of infected farms. I have, however, in Table VII (C) given data relating to the farms found tuberculous more than once. Mr. Greenwood's criticisms of the constitution of some of my statistical groups are, I believe, in great part based on a misunderstanding of the nature of my data, and of the objects which I had

¹ Reproduced from the *Transactions of the Manchester Statistical Society*, November, 1909.

DIAGRAM.

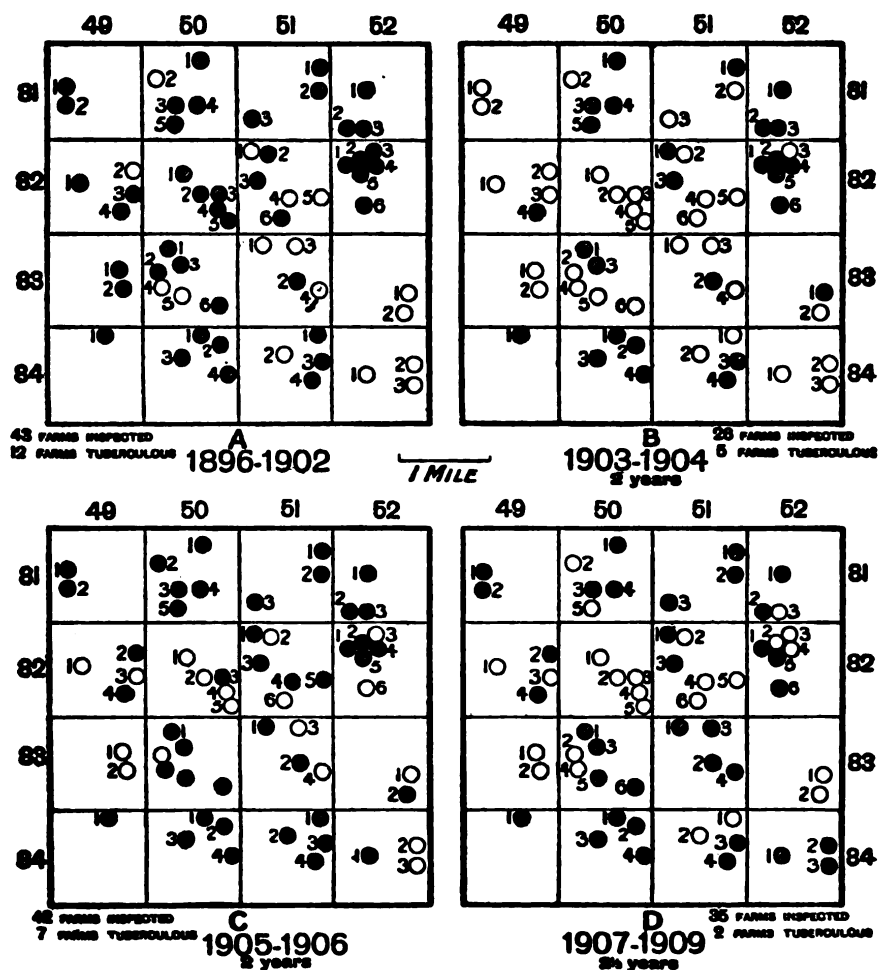
One sixteen-square-mile area (XXI, M).

Showing the results of the inspection of the farms supplying milk to Manchester during the fourteen years 1896-1909 (inclusive). Divided into four periods: A—1896-1902; B—1903 and 1904; C—1905 and 1906; D—1907.

The position of the farms is indicated by a numbered ring. ○

The farms inspected during each period are tinted grey. ●

The farms inspected and found tuberculous are black. ●



N.B.—The farms found tuberculous in the earlier periods, and not re-examined in subsequent years, had ceased sending milk to the town.

in view. I have not stated in any part of my paper that altitude had no effect upon the prevalence of tuberculosis. I have only said that the effects indicated by the altitude data at my disposal were insufficient to explain the differences observed between various districts. The utmost difference between the farms situated below the 300 ft. line and those at 300 ft. or more is that indicated by the difference between 26·2 per cent. for the first and 19·2 per cent. for the second group. The regrouping suggested by Mr. Greenwood for the purpose of showing that altitude has a distinct effect reduces the difference to one between 25·2 per cent. and 19·5 per cent. The differences brought out by either method are small when compared with that between 10·5 and 29·7, which are the percentages obtained with regard to certain districts north and other districts south of Manchester irrespective of altitude. Moreover, the differences between the maxima and minima in each altitude group are so great that one is led to doubt the practical value of the averages, and this difficulty remains whatever method of grouping is adopted. Therefore, although the figures suggest that altitude within the limits in question has some effect, I maintain that the figures do not give any support to the supposition that this effect is sufficient to account for the inequalities of prevalence in various localities. Mr. Greenwood's references to Table VII were influenced by his belief that the figures given in the last column had a rigidity which they have not. If he refers to Table V and to the averages given in Table VII for all the non-tuberculous farms and all the tuberculous farms respectively in regions north and south of Manchester, he will find that a clear difference in favour of the farms well managed is indicated. The figures in the last column but one on the right (Table VII) which have attracted specially his attention refer only to the number of tuberculous udders which have actually been found on the infected farms by the veterinary surgeon. I say clearly in my text that little importance should be attached to small differences in these percentages, because the data upon which they are based are only approximately comparable. The application of rigid statistical methods to data of this kind would not only be a waste of time, but might lead to very erroneous conclusions. If, apart from the keeping of infective animals, the general management of the farm had had a dominant influence upon the prevalence of tuberculosis, this would have been indicated by differences in the percentages so marked that the errors I have alluded to could not have obliterated them, for the differences which have to be accounted for are, as I have shown, very large. The approximate uniformity of the percentages obtained with regard to groups of farms which are so differently managed shows that something else than general management, including state and size of buildings, ventilation, cleanliness, general condition of stock, &c., is the dominant factor, but this does not prove that management is without effect.

I quite agree with Dr. Buchanan that it will be impossible to obtain quite satisfactory statistics regarding the distribution of bovine tuberculosis

until every farm and cow within a definite area have been thoroughly inspected and tested. This is a view which I have myself advanced on more than one occasion since the year 1892, but as this would involve the existence of power and means of inspection which do not exist at the present time, and which may not be available for some time to come, it appeared to me that my facts, incomplete as they are, might, in the absence of more satisfactory information, prove useful. The question of the possible influence of a tuberculous farm upon neighbouring farms is one which at one time appeared to me to be of importance. Some of my early observations upon the grouping of tuberculous farms had led me to believe that there was some evidence of farm-to-farm infection. Under that impression I made inquiries to discover how such an infection could take place. I found that there was usually very little exchange of cattle, fodder, manure, &c., between adjacent farms, and that the use of common grazing fields was very unusual. But as my data accumulated I realized that even in the worst districts there were groups of farms which remained for years unaffected, although they were in the vicinity of farms severely affected; and I finally came to the conclusion that although infection from farm to farm is not impossible, it is not of common occurrence. A connexion between non-tuberculous farms and neighbouring tuberculous farms has on several occasions been clearly brought out with regard to the supply of tuberculous milk, but this was due to the fact that some farmers who had run short of milk had made up the deficiency by obtaining milk from a neighbour who happened to keep a cow suffering from tuberculosis of the udder. On more than one occasion a non-tuberculous farm has been found to send to Manchester tuberculous milk which, on investigation, was traced to a neighbouring tuberculous farm. This indicates one of the possible ways in which a non-tuberculous farm might be infected by a tuberculous farm. Dr. Buchanan suggests that the proportion of tuberculous milk sent from a certain district to Manchester and some other towns respectively might be fortuitous, and that there was nothing to show that the farms sending milk to the two were the same or comparable. Chance could certainly not have been excluded if I had dealt with a single district supplying two towns only, but by referring to Table IV in my paper Dr. Buchanan will see that the results recorded have a more general character. In the first instance the facts relate to three perfectly distinct districts all fairly limited. The northern district supplied Manchester and town A. The eastern district supplied Manchester and towns B and C. The southern district supplied Manchester and town D. The tests were all made during a period of two years; each sample corresponded to one farm on which there were on an average from sixteen to twenty cows. The number of samples tested is given in the table; the smallest number relating to one town was forty-nine, corresponding to not less than 700 to 800 cows. The farms supplying Manchester and the four other towns were all different, but had that in common that they were respectively situated in one of the three selected areas. All the samples

were collected according to my instructions and examined in my laboratory. Chance can hardly be supposed to account for the following facts :—

(1) Each of the four towns received during the two years a larger proportion of tuberculous milk than Manchester.

(2) The proportion of tuberculous milk sent from the eastern district to Towns B and C was in each case much greater than the proportion of tuberculous milk sent to Manchester.

(3) The proportion of tuberculous milk reaching Manchester from each of the same districts bore a distinct relation to the amount of tuberculous milk supplied by these districts respectively to the four other towns.

I think it most improbable that such a series of concordant facts could be attributed to accident. It would be very interesting to know, as Professor Simpson suggests, the history of the cows on the several farms. It is, however, obvious that the task of keeping a record of the history of some 30,000 cows supplying Manchester with milk is beyond the range of possibility at present. Some interesting cases have been kept under observation to study the mode of development of tuberculosis of the udder. In two or three cases evidence of healing has been observed, but in most cases of tuberculosis of the udder the history of the cow has been short, for, whenever possible, the animals have been slaughtered as soon as practicable.

Dr. Meredith Richards raises the question of the value of clinical examination for the purpose of eliminating tuberculous cows. This question is not within the scope of my paper. I may, however, quote some of the results given in my Report to the Local Government Board (p. 413). Out of 940 udders which, *on clinical grounds*, it seemed desirable to examine for tuberculosis, only 242 were proved by bacteriological examination to be actually tuberculous. It is only fair to say that some of these udders showed very doubtful clinical evidence of disease, and were tested only to gain information. These figures give, therefore, a rather exaggerated idea of the failure of clinical methods; but I think I do not exaggerate when I say that not more than one third of the udders which had characters suggesting the possibility of tuberculosis have been found to be actually tuberculous. With regard to tuberculosis of internal organs, a comparison between the results obtained by clinical examination and by tuberculin testing (confirmed by post-mortem examination) shows that the proportion of tuberculous cows that escape detection, when the clinical method alone is used, is very considerable. For administrative purposes the clinical method is quite insufficient and unreliable, and if adopted generally would give rise to serious difficulties.

As Dr. Coutts points out, in a certain number of cases the veterinary inspectors have not been able to discover a cow with a tuberculous udder among herds that had supplied tuberculous milk. I was at first under the impression that milk might be fairly frequently infected by tuberculous

discharges from the intestine or genito-urinary passages, and sometimes also by other tuberculous discharges, fresh or dry. These sources of infection do not, however, appear to play a very important part. I have dealt with that point in detail in my report to the Local Government Board, and have found that when nothing interfered with the work of the veterinary inspector he was able to detect one or more cows with tuberculous udders in nearly every herd supplying tuberculous milk. An analysis of the results of the examination of 7,000 samples of milk showed that the infection of the milk was due to—

Tuberculosis of the udder in	78·6 per cent. of the farms
Probably to tuberculosis of the udder in	16·0 „ „
To various causes, including undetected cases of tuberculous mastitis in	5·2 „ „

In winding up the discussion Dr. Niven has very clearly indicated some of the most important issues raised in my communication. He pointed out that there was coincidence between common-sense reflection and my conclusions. It is a sign of the progress which has been made of recent years that it should be possible to express such an opinion, because at the time when my investigations were started, and for many years after, so much importance was attached to heredity, breed, housing, fresh air, feeding, &c., that the importance of the actual source of infection was at times almost overlooked. Even at the present time I doubt whether there would be many agriculturists who would be prepared to accept the views I am defending. Although, therefore, I am in perfect agreement with Dr. Niven, Dr. Martin, and Mr. Greenwood as to the importance of management of the farm, I would like to differentiate the elements of good management. Under management I presume that we must include everything that depends on the knowledge, activities, and care of the farmer, such as state of the sheds, ventilation, cleanliness, feeding, selection and disposal of stock, and prevention of disease. I have shown that the situation of the farm, including its altitude—which is beyond the control of the farmer—has not a paramount influence upon the prevalence of tuberculosis. I have attempted to find which of the various elements that have to be considered in the management of the farm was the factor that had the greatest influence on the occurrence of tuberculosis, and I have come to the conclusion that this factor was *the cow in the actively infective state of tuberculosis*. In other words, although sources of tuberculous infection are probably present in most farms, these are fairly easily kept under control by ordinary methods of management; but if management does not include measures having for object the keeping of herds free from the presence of animals in a state of advanced tuberculosis, the best management fails to prevent infection. At present, good management as understood by most of the farmers and many of the authorities includes everything *except* :—

(1) The precautions necessary to avoid the introduction of diseased animals into the herd.

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(2) The periodical renewal of stock so as to avoid the accumulation of cows having reached the age at which tuberculosis becomes a serious source of loss and infection.

(3) The periodical inspection of herds so as to remove and isolate without delay all animals that might become a serious source of infection, and the adoption of other measures necessary to remove or sterilize infective matter.

If the thorough measures which I have invariably advocated for the stamping out of bovine tuberculosis were adopted, much of this work would be done automatically, under the supervision of the state, by local authorities; but so long as matters remain in the present state the farmer must be induced to exercise private initiative with or without the assistance of local authorities. The chief argument of my thesis is therefore *that massive infection due to actively infective cases of tuberculosis constitutes the dominant factor determining the incidence of tuberculosis.*

Epidemiological Section.

May 27, 1910.

Dr. JAMES NIVEN, President of the Section, in the Chair.

Discussion on the President's paper on "Summer Diarrhœa and Enteric Fever."¹

The PRESIDENT reminded the meeting that it was a paper which dealt exclusively with the transmission of disease by flies, and the consequent presumed effect on the summer wave of diarrhœa. Epidemic waves of disease were affected by various influences, amongst others by transmission, and presumably by transmission by insects. They were affected by conditions of weather, no doubt acting indirectly on the system. The indirect effect of such influences on the human system could not be entirely ignored in any instance. For many years he had thought that the effect of such influences, especially on the chemical constitution of the blood, must be of very great importance in connexion with varying periods of epidemic waves. Notwithstanding that fact, and that measles and other diseases had different periods from summer diarrhœa, he thought a case had been presented showing the probability that the wave of summer diarrhœa was due to transmission by flies.

Dr. COPEMAN said he was glad of the opportunity of opening the discussion, as it provided the occasion to offer the President the best thanks of the Section for his valuable and interesting paper—a paper which, by reason of the records of accurate observations carried out over a long series of years, might truly be called a monumental work. For some time past he had himself been engaged, under the instructions of the Local Government Board, in directing an investigation into the subject of the possible carriage of infection by flies. But on the point with which Dr. Niven mainly dealt—namely, the parallelism between the incidence of flies and of summer diarrhœa and enteric fever—

¹ Read on April 7, 1910 (*see* p. 131).

useful work had not proved possible, mainly because of the unfavourable meteorological conditions, particularly the low temperature and heavy rainfall which had prevailed during the last two years. With regard, however, to the other points dealt with in the paper, he and his colleagues had carried out some work, certain of the results of which had already been published in official reports of the Medical Department of the Board; while a further instalment would follow shortly. Dr. Niven had referred to the inquiries which had been carried out in former years, notably by Ballard, Newsholme, Delépine, and others. Ballard's work definitely established the relationship of the commencement of incidence of summer diarrhœa with the temperature recorded at a point about 4 ft. below the surface of the earth. He did not suppose people now held the opinion that this correlation was due to the growth of a specific organism in the soil at that depth, which subsequently reached the surface and then became wafted in the air. But that this curious relationship between the earth temperature and the appearance of epidemic enteritis actually existed was an undoubted fact, the precise significance of which was dealt with in the paper. The work of Newsholme and Delépine dealt largely with milk infection as bearing on the appearance of summer diarrhœa, though they apparently arrived at somewhat different conclusions. Professor Delépine concluded that the infection caused in the milk was due to it having been exposed to high temperatures during long transit from where it was produced to the consumer. But Dr. Newsholme believed the infection was entirely domestic. Certainly the infection of milk supplies had much to do with the incidence of epidemic enteritis, and the domestic view seemed the more likely. Dr. Niven's first thesis was that the disease called summer diarrhœa was a definite entity and an infectious disease, and that was not likely to be cavilled at. It was by general consent regarded as an infectious disease, and for this reason the College of Physicians had termed the disease epidemic or zymotic enteritis. Cases of so-called summer diarrhœa had indeed a symptomatology which was practically identical in every instance. He did not, however, think that at the present day there were any means other than clinical of deciding whether a case should be regarded as summer diarrhœa, and the fact that but little was known as to its causation from a bacteriological point of view made it difficult to state anything dogmatically as to its possible causation by flies. From time to time all sorts of micro-organisms had been credited with the causation of summer diarrhœa. Dr. Klein had suggested that *Bacillus sporogenes* was the infective agent; others that Gaertner's bacillus was concerned in the matter. But probably the most important work on the subject was that done recently by Dr. Morgan and his colleagues at the Lister Institute. They had isolated and worked with a micro-organism which they believed to be the specific cause of the disease, one belonging to the same class as the others to which he had referred, and all somewhat related to the *Bacillus coli*. When the bacteriology of summer

diarrhoea was better understood, it would be possible to estimate more satisfactorily the relationship of the disease to the prevalence of flies. With regard to enteric fever, they were in a better case, as it was usually possible to decide in an early stage whether the disease was actually enteric fever or not. There were two methods available for diagnostic purposes: either Widal's test, or the method of removing a small quantity of blood and inoculating it into nutrient material containing bile salts. He hoped before long there would exist equal facilities for determining whether a definite series of symptoms were those of enteritis or not. In his paper (p. 134) Dr. Niven referred to the well-recognized fact that in Manchester, as in other towns, diarrhoea mortality was very high in all the districts in which privy-middens were prevalent, and the author stated that as supporting the thesis that the fly was concerned in the disease. Although the small house fly never appeared in such quantities as the larger house fly, it not improbably played more than a subsidiary part. The author referred to the social conditions which played a part in the disease, and referred to the work of Dr. Hope and other observers on the question as to artificially-fed infants suffering more from fatal diarrhoea in the first three months of life than did breast-fed infants. The conclusions drawn in the paper were admirably supported by the series of curves of annual incidence of flies and of summer diarrhoea which the President showed at the last meeting. The question was to determine how far the parallelism bore out the contention that the one was dependent on the other. He (the speaker) confessed that in his opinion there was a definite relationship between the two, and he had come across an interesting example of that in the course of an official inspection of Wigan. That town was a privy-midden and pail town. In St. Patrick's Ward lived the lowest class Irish population, and it was in that ward that nineteen years ago he had had to investigate the circumstances of a large outbreak of typhus fever. On revisiting the town a year or two ago he had paid special attention to the incidence of summer diarrhoea, and he found it was not that very insanitary area which had the heaviest incidence. That was found in a small triangular area cut off from the rest of the town by railways, on two sides of an isosceles triangle, the base of which was formed by the municipal depot to which the pail contents were taken and stored in large tanks prior to removal, at the convenience of farmers, by barges on the canal. The houses were good and the people above the average of those inhabiting St. Patrick's Ward, and the only reason to which he could attribute the special incidence of summer diarrhoea was the prevalence of flies due in turn to the enormous accumulation of excreta. The people told him that in hot weather the flies swarmed in at their doors and windows and all over their food. He thought the relationship there was undoubtedly one of cause and effect. He would leave the records which Dr. Niven showed on the last occasion to be discussed by Dr. Hamer, who had been engaged on similar work for the London County Council. He (Dr. Copeman) would confine

his further remarks to the methods of research being adopted in that kind of work.

On p. 141 Dr. Niven referred to his own methods, especially the use of the beer-trap, from which the captured flies were afterwards emptied out and counted. He (the speaker) did not regard that trap as so useful as other methods. Perhaps as good results would be found by such means as those employed by Dr. Hamer, particularly the use of fly-papers and "balloon" traps. Blue-bottles and green-bottles were sufficiently powerful to get free from the sticky papers, so one should use the "balloon" traps in addition, so as to make certain of the relative numbers of the different species of flies. As bait for the traps, he used coarse brown sugar dissolved in a little stale beer. Dr. Hamer had shown that there was a curious and definite distribution of flies on papers which enabled one to distinguish more readily than otherwise the *Musca domestica* from the *homalomyia*. The two kinds of flies attached themselves to different portions of the paper. With regard to the breeding of flies, and the manner in which incidence of flies was carried over from one year to another, little was known of this until recently. On p. 155 Dr. Niven referred to the experiments of Griffith, and he (the speaker) would call attention to a paper by Jepson, in the first of the two reports issued by the Local Government Board, in which it was shown that under favourable circumstances it was possible for flies not only to persist in an active condition, but to breed during the winter months, and in that way flies could be carried over from one fly season to the other. There could also be no doubt that flies hibernated, and so could pass the winter in protected situations. He had had some correspondence with Dr. Laver, of Colchester, a good antiquarian and naturalist, who informed him that he had found that the upper part of a stack, just under the thatch, was a favourable place for the hibernation of flies. They certainly also hibernated in houses, even though they might not be visible for the time, to come out in the first days of spring and form the progenitors of the summer broods. The author said on p. 155 "there is little or no doubt, in fact, that the seasons are connected by living flies and not by pupæ." He (the speaker) would be glad to know if Dr. Niven had any observations bearing on that statement. An important matter in connexion with possible carriage of infection was the range of flight, in an upward and a horizontal direction, of flies. Vertical flight would come into play in the case of tenement buildings, and horizontal flight in regard to the distance which the flies might be expected to traverse from one point to another. That had, to some extent, been worked out by Dr. Arnold and others. In some work which had been carried out at Bermondsey he had been able to take advantage of the fact that a green-fly which was present in enormous quantities at the glue works, bred in the bags of putrefying bone brought to the works, and by setting traps at varying distances around the glue works it had proved possible to determine how far the flight of these flies could extend. The greatest distance at which they were captured was

about 200 yards, the place being the kitchen of a County Council Infants' School. If one wanted to trace the flight of the *Musca domestica* and other flies, they must be marked in some way. Dr. Arnold did that by painting a small patch of enamel on the thorax—necessarily a slow and tedious operation. A method of marking flies with the minimum of trouble, devised by Mr. Jepson, consisted in shaking them up in a large paper bag containing finely-powdered red chalk. Thousands of flies could be marked in a few minutes in that way, and the marking persisted for at least three weeks. So arrangements had been made to carry out during the present year a series of experiments on the flight of flies at Cambridge. Stations had been established about 50 yards from one another in a radial direction from the central experimental station. Of course, a fly would travel further with the wind than against it, so that observations of this and other meteorological conditions would be kept, control experiments would also be carried on in a district or the outskirts of Cambridge where there were practically no buildings. Another important point to which Dr. Niven referred concerned the carriage of infection by flies. Flies could carry on the outside of their bodies a certain amount of infectious material. The body of the fly was covered with minute hairs, therefore it was not to be wondered at that bacteria could attach themselves to them. That was shown by Sherrington and himself twenty years ago. In comparatively recent years other people had brought forward work on practically identical lines. But for what length of time could flies carry organisms which might be dangerous? Owing to their being exposed to sun, light, and air, pathogenic organisms were not likely to persist for long on the surfaces of their bodies. A question of more importance was whether the fly was able to convey micro-organisms in the interior of its body. Possibly also protozoa might have some relation to the carriage of summer diarrhoea in regard to food contamination. The fly could not take up solid material, and it must moisten a grain of sugar before it could feed on it. So that if it had any pathogenic micro-organisms in the fluid extracted from its proboscis, they would be likely to be deposited on many grains of sugar while the fly was feeding. In houses, sugar was the last substance to be protected from flies, yet it was largely added to children's milk, which was, of course, a food medium for the multiplication of micro-organisms. In that way the child might become infected. On p. 209 Dr. Niven questioned whether other insects than flies might not have something to do with the carriage of infection. He (the speaker) did not know whether Dr. Niven had any definite evidence about cockroaches, but he did not know why the author made the dogmatic statement as to fleas and bugs playing no part in the process. In a recent official report, he (Dr. Copeman) had committed himself to the suggestion that it was possible that fleas might have had something to do with the spread of the infection of enteric fever. Reference was made particularly to the circumstances of an outbreak of enteric fever at Workington. It was an outbreak which occurred among the poor population in winter-time, when,

owing to the shutting down of the iron works, there was not much work. In order to save rent, people crowded together into houses; 90 per cent. of the cases admitted to hospital bore evidences of flea-bites, and rose spots subsequently appeared around the bites. Flies were, in all probability, absolutely useless in our present stage of civilization, and therefore every effort should be made to get rid of them. He was sure they played a definite part in the carriage of disease. First, their breeding must be prevented, the means of doing which were obvious. Their larvæ should be destroyed by disinfectants; the adult insects by traps and poisons. Possibly also some use might be made of cultivations of their natural enemy, the fungus *Empusa muscæ*. If by reducing the number of flies one could diminish the amount of summer diarrhœa and enteric fever, a great advance would have been made in sanitary work.

Lieut.-Col. A. M. DAVIES: In the last Army Medical Report for 1908-9 a close connexion between prevalence of flies and of enteric fever had been recorded in many instances. At Sabathu thirteen cases occurred in May; flies were stated to have been "a perfect plague" in April and May, being most numerous in the cookhouses. At Jubbulpore twenty cases occurred between March 24 and the end of May; just previous to the outbreak, and during its early part, flies were very prevalent. At Poona and Kirkee a study of the admissions for enteric during fourteen years shows that with great regularity enteric reaches its maximum in August and September; in July "extraordinary swarms of flies" are present, and sometimes remain until October. Captain Ainsworth exposed sheets of "tanglefoot" in three kitchens for fixed periods, afterwards counting the number of flies caught. Up to the middle of June the numbers were small, 300 or less caught daily; towards the end of June they increased, 550 to 700 daily; in the middle of July, 900; week ending July 24, 1,150 daily; July 31, 700; August 7, 150. The cases of enteric began to appear at the beginning of July. In the week ending July 31 there were eight admissions; August 7, six admissions; August 14, ten; August 21, eight; then they decreased. Fly prevalence certainly was followed by enteric prevalence. At Secunderabad forty-four enteric cases occurred in July, August, and September; for some time preceding there had been a regular plague of flies. At Ahmednagar there was noted a very instructive outbreak of ten cases of enteric. Previously there had been no enteric admissions for four months; then two companies of the Worcester Regiment were moved into the Artillery lines, where the latrine accommodation was insufficient and the sanitary establishment not equal to the increased work. As a result, removal of excreta was not properly carried out, the latrines became fouled, and flies swarmed. The use of a disinfectant solution in the latrine pans, which had been in force for a year, was for some reason discontinued, dry earth used instead, and the flies returned. After a short time measures were taken and a fly campaign started. The outbreak was confined to these lines, and ceased twenty-three

days after its commencement. At this station the latrines were only 40 ft. from the cookhouses, and contamination of food by flies was very likely, as the speaker had found to be the case ten years ago. At Mhow also, which has a bad reputation for enteric, a campaign against flies was undertaken in 1908; rubbish and litter and other breeding-places were cleared away, and the results were most satisfactory; the cavalry regiments, which used to spend sixty rupees a month on fly papers, were able to discontinue their use. These Indian instances are all in favour of a connexion between fly prevalence and enteric fever; when a campaign has been carried out against flies, by thorough regard for cleanliness and prevention of fly access to excreta, enteric has diminished or disappeared. As to the question whether the fly carries the infection on its surface or (through feeding on infective matter) in its excrement, the speaker thought that it was probably through flies gaining access to specifically contaminated excreta, on which they fed (as pointed out by Major Faichnie), that the spread of enteric by flies was chiefly brought about. Some years ago drinking water was considered to be the chief medium of spread of enteric; at present "carriers" were thought to be of more consequence. Certainly in India water was not the only, nor the chief, mode of dissemination; carrier cases probably were responsible for a good deal. But no one could doubt that spread by means of flies was an extremely likely occurrence in that country, where latrines and cookhouses were often very near together, and there could be no question but that flies went to and fro between them constantly. Measures have been taken within the last few years to prevent this.

Col. NOTTER said the greatest incidence of enteric fever in India was at the drying-up of the rains—more so then than during the rainfall. There was no doubt about the influence of flies in spreading this disease. That was clearly brought out in a report on dysentery in South Africa which was published in 1900. The water supply, in many cases, was as pure as it could well be—it was brought from a pure source—yet the number of cases of enteric was very large. At Pretoria it was no doubt due to flies; there was there a system of pail conservancy, the pails being emptied in a haphazard manner and taken two or three miles outside the town. There were many officers and non-commissioned officers who contracted the disease, both in the town and in camps. There could be no doubt that flies were carriers of infection, and it was chiefly by them that this disease was spread.

Dr. HAMER said: Dr. Niven had, in this paper, brought to a focus observations made during the last seven years, during which time he had led the way in investigating the part played by flies in connexion with typhoid fever and diarrhoea. He had throughout this period been ever the first to recognize difficulties, the first also to suggest the solution of these difficulties. It was a disappointment, to those who were still sceptically minded, to find him now, after his exhaustive examination of the potentialities of the fly-

bacillus-mechanism, disposed to attach so considerable a degree of importance to it. After all, the foundations upon which the fly theory could rest had still to be laid down. The suggested mechanism, it was true, could be fitted to certain observations; but he (Dr. Hamer) could not help recalling the warning "so long as a suggested mechanism accounts only for the phenomena which gave rise to it, it can only claim to be a possible solution of the riddle. It is when a mechanism is found to account also for other phenomena, widely different from those that suggested it, that it first becomes entitled to claim to be regarded as the actual solution." As a preliminary test it was worth considering one or two subsidiary supporting propositions for which the theory might be held accountable, or which were themselves necessary to the theory.

For example, is summer diarrhœa an infective disease? Dr. Copeman had appealed to the nomenclature of the Royal College of Physicians; but that was subject to periodical revision. The view generally held, until a few years ago, was that expressed by Dr. Niven himself in his annual report for 1898: "The disease is not an infectious one; that is to say, its occurrence is not usually, indeed rarely, marked by simultaneous or successive attacks in the same family." Then followed his inquiries of 1904 and 1905. As to the lessons to be learnt from these, and from the similar investigation of Dr. Sandilands, there was apparently room for difference of opinion. Dr. Sandilands expressed himself with much caution. He endeavoured, moreover, to obtain evidence from institutions. Dr. Niven rather agreed that further inquiry as to institutions should be made. Now, however, he said (p. 133): "Epidemic diarrhœa is an infectious disease. If the history of institutions were faithfully recorded, it would probably be found that it not infrequently spreads in these." But this was counting the chickens before they were hatched.

Again, there was the question of the relation of stables, privies, &c., to diarrhœa. Dr. Niven referred (p. 134) to the inquiries of Boobbyer, Scurfield, Tattersall, and others. But he, himself, had been the great explorer in this difficult field. He had dealt with complicating circumstances, such as those introduced by high birth-rates, social conditions, &c. (Annual Report, 1898). He had shown that some midden districts in Manchester were hard hit in years of high prevalence of diarrhœa, while others were not. "The facts," he said, "lend only a general support to the theory." And, further: "We have not gained so much from that line of argument as we might have hoped; but, nevertheless, the table forms an important part of our reasoning as regards infection." Again (p. 186) the sudden fall of typhoid in Manchester in 1899 was mentioned as constituting a difficulty, for the drop "was not due to conversions [of privies into water-closets] which are now proceeding rapidly, but were not in progress at that period." Finally (p. 187), there was the inconstancy of appearance, in different years, of the seasonal variation in the proportion of cases of typhoid occurring in midden houses. The more closely this question of middens was looked into, the more perplexing it proved to be.

A third supporting proposition which might be mentioned was the thesis that one attack of diarrhoea protects. There, again, evidence was needed. Dr. Sidney Davies's Woolwich figures, at any rate, lent no support.

Dr. Copeman had stated that he did not attach much importance to the fact that if a fly is made to walk on solid nutrient material, growths occur on the line of its track. All would agree, and be at one in feeling, that the main point of interest in connexion with Dr. Niven's paper was to determine whether it furnished any independent epidemiological support to the ordinary laboratory argument. Dr. Niven, it might be noted in passing, assumed (pp. 138 and 139) that the growth of the causal organisms in question in this paper was "confined to a comparatively short period, corresponding to the periods occupied in the production of laboratory cultures." This was a large assumption (it was only necessary to refer to Dr. Peters's paper read before this Section¹). A further step, however, was that taken on p. 140. By a process of exclusion merely, it was now inferred that "what we require for the explanation of the facts of summer diarrhoea is the presence of some transmitting agent, rising and falling with the rise and fall of diarrhoea." A famous scientist was said to have warned his pupils that, in scientific research, "unless great care was exercised, there was always the danger that one would find what one was looking for." They certainly seemed, on reaching p. 140, to be incurring this risk. And, beyond this, even after the epidemiological inquiry was completed, it was not altogether clear (*see* p. 206) that they did not end, as they began, with the bacteriological argument. Dr. Niven had, however, called attention to ripples on the swell of the rising typhoid wave. It was true his explanation removed some difficulties, but it introduced others as great as those removed.

Thus two main statistical anomalies had hitherto been encountered in this connexion. Both were originally pointed out by Dr. Niven. The first he thus referred to (Annual Report, 1904): "It is noticeable that the descent in the number of deaths precedes the diminution in the number of flies. It is possible that this is due to exhaustion of material. Now, an exhaustion hypothesis was hard to accept; it was clearly insufficient, at any rate, in the years of low incidence. The aid of the fly fungus in immobilizing the fly was therefore invoked. The process, judging by the diarrhoeal curve, must be in full operation from the thirtieth to the thirty-fifth week; but from the thirty-seventh to the forty-first week (i.e., some weeks later) came the secondary wave of typhoid fever (p. 191) "associated with flies in the manner indicated." Inasmuch as the flies could not be both immobilized and active at one and the same time, if the fly-fungus explanation were relied upon for diarrhoea, the fly causation of the secondary wave of typhoid could not, it would seem, be adhered to.

¹ *Proc. Roy. Soc. Med.*, 1909, ii (Epid. Sect.), p. 1.

The second difficulty was the old one with regard to absence of delay in the appearance and disappearance of diarrhoea, for the diarrhoea and fly curves were practically superimposed. Thus Dr. Niven said (Annual Report, 1905): "The impression produced by the two sets of figures in 1905 is rather one of simultaneity than of cause and effect; and if we take the figures as indicating that flies do cause the rise in the number of cases, we must also assume that their action is very rapid." It was this last assumption that was the source of perplexity, for it must be remembered that there were two things to be accounted for—the suddenness of the rise on the one hand; the alleged case-to-case infectivity on the other. When the fly-bacillus mechanism was geared for rapid work—the production of the "sudden uprush,"—minutes, or at most hours, were in question; but when fixing the period to be allowed for tracing infection back to its source, it became a question of weeks. Might it not be argued that the average time allowed for transmission of infection must be the same in both instances?

The position in the case of diarrhoea was peculiar. In some infections—those, for example, which tubercle or typhoid bacilli were believed to originate—they were not hampered by the question of rapidity of transmission, and as a matter of fact there were those who did not hesitate to go back thirty or forty years to meet the requirements thought to be necessary. In the inquiry into diarrhoeal infection of 1904, Dr. Niven made no extravagant claim of this sort. He only once went back—and that in a doubtful case—to a previous summer; in a number of instances his interval was under a week; in others it was between a week and a month; in six cases it was a month or over; the average time was between two and three weeks, an interval which, it was important to note, compared with that Dr. Niven himself (p. 205) allowed for delay in transmission (apart, of course, from any question of length of incubation) in typhoid fever. But this average interval, while found to be necessary in a disease proceeding in orderly fashion by primary and secondary waves, did not accord with the "sudden uprush" of diarrhoea. Dr. Niven agreed, for he said (in 1904) of his thirty-three cases of direct infection: "They do not furnish any explanation of the manner in which the curve of diarrhoea shoots up in summer." And yet the cases he was discussing occurred in the height of summer, in rooms infested with flies. If flies ever operate with great expedition, the conditions in nine out of every ten of the households in which the inquiries of 1904 were made, where flies were "numerous," or in "tremendous numbers," or in "swarms," were such as to demonstrate the fact. It might conceivably be urged that the three weeks' interval was too long, and that the average time for transmission of infection was only a day or two. If that view were accepted, the thirty-nine cases would be reduced almost to vanishing point, and diarrhoea, so far as evidence based upon multiple cases in houses was concerned, ceased to be conveyed directly from case to case by flies or by anything else.

Dr. Niven said (p. 181): "If flies are capable of conveying infection from one house to another, they are, *a priori*, capable of transmitting it within the house." Already, in 1905, however, he had recognized the difficulty with regard to these cases within houses, and he wrote: "Apparently house after house is invaded in rapid succession, the infants being attacked without any history of previous diarrhoea to be obtained in the older members of the household." He sought for evidence of fly infection in "nests," and studied the circumstances of two areas in this connexion. But in the two areas, as in the thirty-nine houses, there was absence of simultaneous or nearly simultaneous attack; and this spreading out of cases made it difficult to reconcile case-to-case infection, however accomplished, whether by flies or otherwise, with the "sudden up-rush" of diarrhoea. If, in fact, the "fly bacillus" or other mechanism linked case with case in the manner supposed, the intervals between rise, attainment of maxima, &c., of the fly and diarrhoea curves must necessarily be substantial ones. This seemed clear, *a priori*, but the need was emphasized by the results of Dr. Niven's and Dr. Sandilands' inquiries, and it could not be gainsaid, if it should prove that Dr. Niven was right in asking for substantial intervals of five or six weeks—three of which (p. 205) he allowed for delay in transmission—in typhoid fever. The question as to the appearance at the times specified of the "primary" and "secondary" waves of typhoid fever was a very difficult one. In 1893, 1896, 1898, 1903, and 1904 the primary wave was absent. With regard to the phenomenon of the appearance of the primary waves sooner (relative to the diarrhoeal wave) in the earlier than in the later years, 1893 and again 1904 and 1907 were conspicuous exceptions. It was important to note that the later years, taken as a whole, exhibited later development of both diseases than did the earlier ones, and, as Dr. Peters had shown, the date of harvest varied correspondingly.

A further point, suggested by the diagrams (pp. 192-9), had relation to the striking differences in the amplitude of the primary and secondary typhoid-fever waves, as compared with that of the waves of diarrhoea, in the several years. In 1891 and 1894 there was much typhoid fever and very little diarrhoea, while in 1897 and 1899 (particularly heavy diarrhoea years) the primary and secondary waves of typhoid fever were but ill marked. Indeed, if 1899 and 1902 were compared, the former, with eight times the diarrhoeal mortality of the latter (and should they conclude eight times the number of flies), had a less marked typhoid wave than the year which Dr. Niven stated (Annual Report, 1904) was one in which the fly was "almost absent" in Manchester.

Other outstanding questions which might have been discussed had time permitted were the "residual increases" of typhoid, which undoubtedly deserved special attention; the reason why typhoid fever in Manchester, and in London also, occurred in "nests"; and the question as to whether there might not be other living (or dead) carriers of typhoid fever in addition to those considered in the present paper.

Dr. PARSONS expressed his appreciation of the amount of labour and observation in the paper. The facts seemed to point clearly to fly infection being one potential cause of epidemic diarrhœa and enteric fever, and also possibly of other diseases, such as cholera and dysentery. But when a new channel of disease was discovered, there was a tendency to neglect the other channels. With regard to simultaneity of attack, which Dr. Hamer thought should occur if fly infection were the cause, it must be rather a matter of chance whether infection was conveyed by a particular fly; not every midden was infected, and not every fly which had visited a midden fed upon food and infected it; it was probably only one here and there out of thousands which did so. Probably, apart from the prevalence of flies, the infection itself was more active and abundant in warm periods, because the organisms flourished best at blood temperature; and that might account for the different effects of dust in March as compared with August. Dr. Meredith Richards made experiments at Croydon, and after diligently watering the streets in the districts where diarrhœa was most prevalent he found a diminution of diarrhœa there. The distribution of diarrhœa and enteric fever did not point to their having precisely the same causes. From 1847 to 1850 the annual death-rate from "fever" in England and Wales per million averaged 1,246; in 1909 it was only 60, or one-twentieth. The diarrhœa mortality had not fallen in the same proportion; it was 1,710 in the first period and 498 in the second, or one-sixth as against one-twentieth. Taking the periods immediately after the passing of the Public Health Act in 1872—namely, 1871 to 1880—and the ten years to 1909—the mortality from enteric fever in the recent period was only 22 per cent. of that in 1871-80; whereas the mortality from diarrhœa in 1900-9 was 53 per cent. of that in 1871-80. During the intervening period there had been a progressive urbanization of the population, although many things had been done to improve public health, with the result that the amount of enteric had fallen greatly coincidently with those improvements. Sanitary engineers had done their best to exclude sewer-gas from houses, and pure water supplies had been provided for all populous communities, and the very success of these measures had led people at the present day to question whether sewer-gas and polluted water had really the importance, as causes of enteric fever, formerly attributed to them. Fly infection probably accounted for part of the enteric fever still remaining. Much had been said about shell-fish causing enteric, but probably that would not apply to diarrhœa, and he did not know whether "carrier cases" could propagate diarrhœa. Susceptibility to diarrhœa was especially present in infants, so the infectiousness would not manifest itself over so wide a range as in enteric.

Diarrhœa was especially a disease of large towns. In 1909 the proportion of deaths from diarrhœa was 0·38 per thousand in the forty-six great towns, and 0·24 in the one hundred and forty-three smaller towns, and 0·17 in the rest of England and Wales. But the proportion of deaths from enteric in each of the

three classes of districts was the same—namely, 0.06 per thousand. So that in the case of diarrhoea there was a cause acting in towns which was not acting in the country. One could hardly suppose that was only flies. He believed enteric did not respond to increased temperature in the same way that diarrhoea did. He knew from letters received from Dr. Ballard that that observer had recognized that the connexion between the prevalence of diarrhoea and earth temperature was not that anything happened at a depth of 4 ft. causing the diarrhoea, but that the 4-ft. temperature was an indication of the algebraic sum of the gains and losses of heat which had occurred during the summer.

Dr. SANDILANDS, in response to the President, said the subject before the Section had already been so fully and so ably discussed that he did not know there was anything he could usefully add. In a certain number of institutions in which he made inquiry there was some evidence of infection, but the evidence did not satisfy him as being conclusive that the disease had spread by infection from case to case.

Dr. WHEATON called attention to the fact that no results of estimations of flies were given for 1907, yet that was a year in which something definite could be found. That year was distinguished by the lateness of the diarrhoea mortality; it attained its maximum on October 12, and continued for some weeks later. The same remark applied in the case of enteric fever. It was unnecessary to deal with privy-middens in this case at all, because there were several large towns having the water-closet system where there was a very high mortality from diarrhoea. With regard to what had been said about the spread of diarrhoea in institutions, he had considerable experience of that, having been physician to a hospital for children and women for many years where there were admitted, as a matter of routine, very bad cases of summer diarrhoea. They never had any suspicion of any spread of disease in that institution, and in his numerous visits to workhouses he had inquired on the point, but had not been able to find evidence that the disease spread in workhouses. Yet many cases of summer diarrhoea were admitted to workhouses, and some workhouses swarmed with flies. He thought there might be some relation between flies and enteric, just as there might be between flies and diarrhoea, but he thought it must be a very limited one. The initial rise, which was such a noteworthy feature in Dr. Niven's charts, might be due to cases of enteric fever suddenly being notified. In a particular district there were certain to be many cases which were regarded by local medical men with suspicion; then one of them notified a case, and instantly others rushed in with notifications of cases about which they had been hesitating for weeks.

Dr. BUCHAN said that at this point of the proceedings there was only time to refer to one point which he desired to bring to the notice of the Section. It referred to the relationship of the curve of flies to the curve of diarrhoea cases.

commencing. They could agree that Dr. Niven had shown that the rise of the curve of diarrhoea cases commencing followed closely the rise of the fly curve in point of time. With the enormous increase in the amount of infective material and number of infective foci, which occurred during the rise, one would expect the beginning of the fall of the fly curve to be succeeded by a rise in the number of cases of diarrhoea commencing; and in those instances, in which appropriate reduction had made the curves closely correspond, it was reasonable to assume that crossing of the curves would take place shortly after the fly curve began to fall. On this point the curves for the years 1904, 1906, and 1908 were against the fly theory, and in the case of the remaining two years, 1905 and 1909, the evidence was not very convincing. He thought, also, that if they examined again all the five curves given of flies and diarrhoea cases commencing in their descent they were bound to state that on the evidence before them the fall in the diarrhoea curve, antecedent to the fall of the curve of flies, was not consistent with the fly theory. The flies represented in the descending part of the curve were active flies in the same sense as those in the ascending part, so far as they had any information. The only evidence of the activity of the flies furnished by Dr. Niven was that they were able to climb into a beer-trap, and they were not supplied with any proof that the flies of the descending part of the curves were affected by *empusa*. A surmise on this point was not, in his opinion, sufficient evidence on which a learned society could support the fly theory of summer diarrhoea. Both the points which he had mentioned appeared to him to be vital, and he begged to put them forward for the consideration of Dr. Niven and the Section.

Dr. DARRA MAIR wrote: Having the data at hand, I thought it might be useful to ascertain whether there has been any similarity between Manchester and Belfast in the curves of weekly deaths from diarrhoea and notifications of enteric fever. Belfast's incidence of enteric fever, it may be remembered, has been so severe that it is without parallel in the United Kingdom, and in the paper I read to the Section last year I endeavoured to show that the only factor which could account not only for the general facts but also for this great excess of the disease was shell-fish—in this case cockles as well as mussels—and that the relationship between privy-middens and rise and fall of fever had been subsidiary. The distribution of the disease excluded water infection. Notwithstanding the excessive mortality from enteric fever in Belfast (in the decennium 1897 to 1906 it was nearly four times that in Manchester), the death-rate from diarrhoea has been appreciably lower in Belfast, approximately as 26 is to 34 per 1,000 births. In the accompanying diagrams (pp. 272-3) the curves of weekly diarrhoea mortality and incidence of enteric fever in Belfast and Manchester during the third quarter (sometimes more) are compared for each year from 1897 to 1908. The Manchester curves have been copied from Dr. Niven's paper, and the Belfast diarrhoea curves have been

made from data in the Registrar-General's weekly returns, the Belfast deaths being divided by $2\frac{1}{2}$, so as to make them comparable with the Manchester deaths, since the latter are divided by 5. It will be observed that the similarity of the diarrhoea curves of the two cities is very close indeed, so close, in fact, that usually the rise of diarrhoea mortality commences in the two cities in the same week, and reaches its maximum either in the same week or in the previous or subsequent week. With few exceptions, too, years of high diarrhoeal mortality in Manchester are years of high diarrhoeal mortality in Belfast, and vice versa, but nearly always the Belfast curve is lower than the Manchester curve. I have also plotted out the mean weekly temperatures of Belfast for the years 1904 to 1908 (as given in the Registrar-General's returns), and these Belfast curves are also practically identical with the corresponding Manchester curves, except, again, that the Belfast temperatures are always lower. I suppose it may be inferred from these data that if flies are responsible for the rise of diarrhoea in Manchester, so also they may be in Belfast, and that the coincidence of the lower range of temperature in Belfast with the lower diarrhoea mortality is consistent with this hypothesis. In no year was the similarity, both of temperature curves and diarrhoea curves, of Belfast and Manchester closer than it was in 1907, when, instead of the diarrhoea maximum being, as usual, before the thirty-ninth week, it did not occur until the forty-first week. Data as to flies in Manchester would have been specially interesting in that year, for if the correspondence between flies and diarrhoea deaths had then been close, it would have been worth a good deal more than the correspondence in the more normal years. Dr. Niven's hypothesis does not appear, necessarily, to involve a relationship between privy-middens and diarrhoea mortality. At any rate, the important factor is the increase in the *number* of flies. In this connexion the experience of Belfast seems important. In Belfast there has been an enormous and rapid reduction of privies, commencing in 1900, and now practically all are abolished in favour of water-closets. There has been, also, an enormous reduction of enteric fever, though the facts, as set forth in my paper, are very strong that the association of this decline of fever with removal of privies has been accidental, or, at all events, subsidiary. But, on the other hand, there has been little or no reduction in mortality from diarrhoea. In the five years 1897 to 1901 diarrhoea mortality amounted to about 28 per 1,000 births, while in the next quinquennium, 1902 to 1906, by about the middle of which the work of privy removal had approached completion, diarrhoea mortality amounted to about 25 per 1,000 births. Moreover, in the summer quarters of 1905 and 1906, when few privies were left, diarrhoea mortality was higher than it had been in 1898, and almost as high as in 1897, when, relatively, privy-middens were nearly universal in Belfast. As in other years, the corresponding incidences of diarrhoea in Manchester were very similar, although in Manchester, as I understand, privies have not been reduced in number to

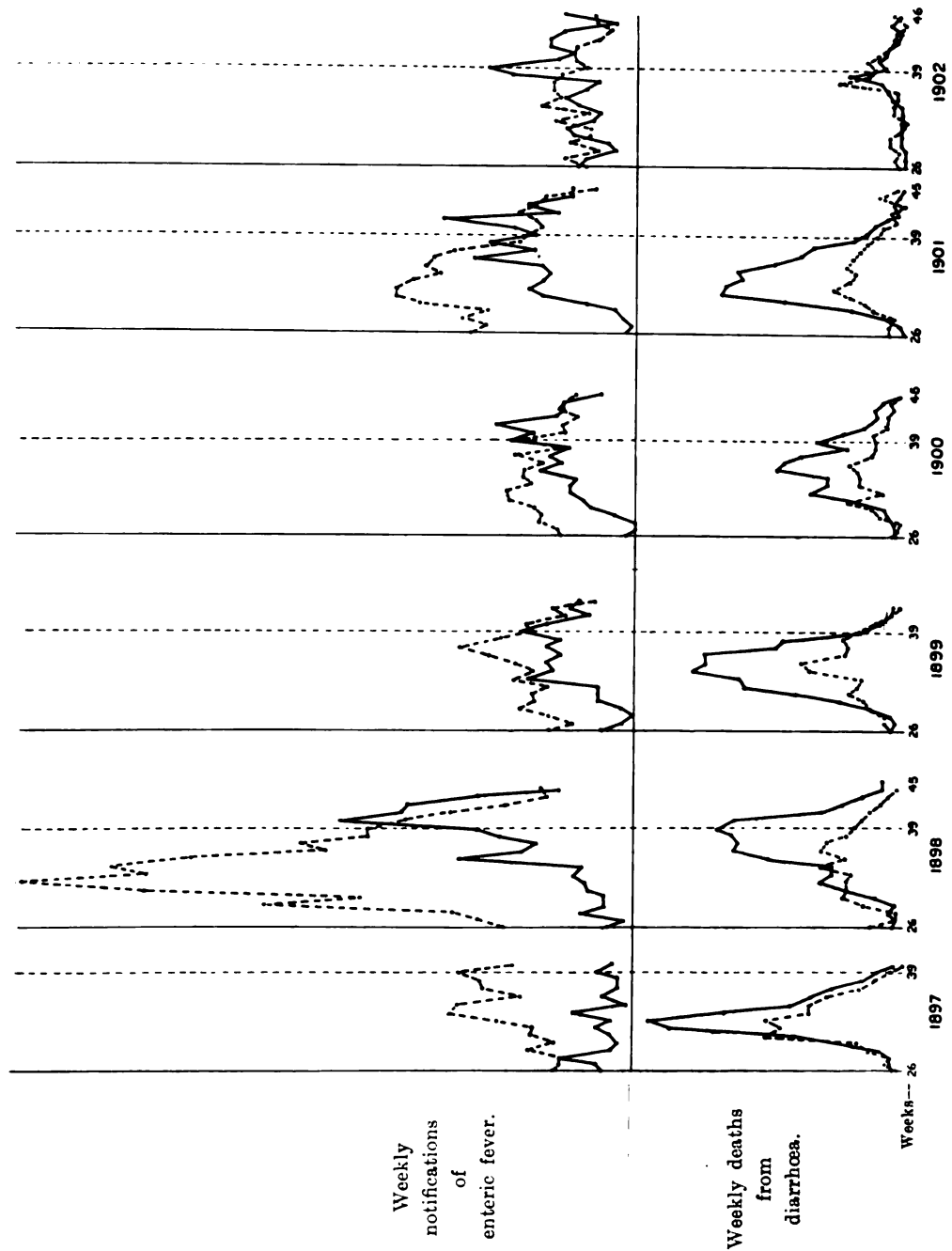


CHART I.—Diarrhoea and enteric fever in Manchester (continuous line) and Belfast (interrupted line) during third and part of fourth quarters in the years 1897-1902.

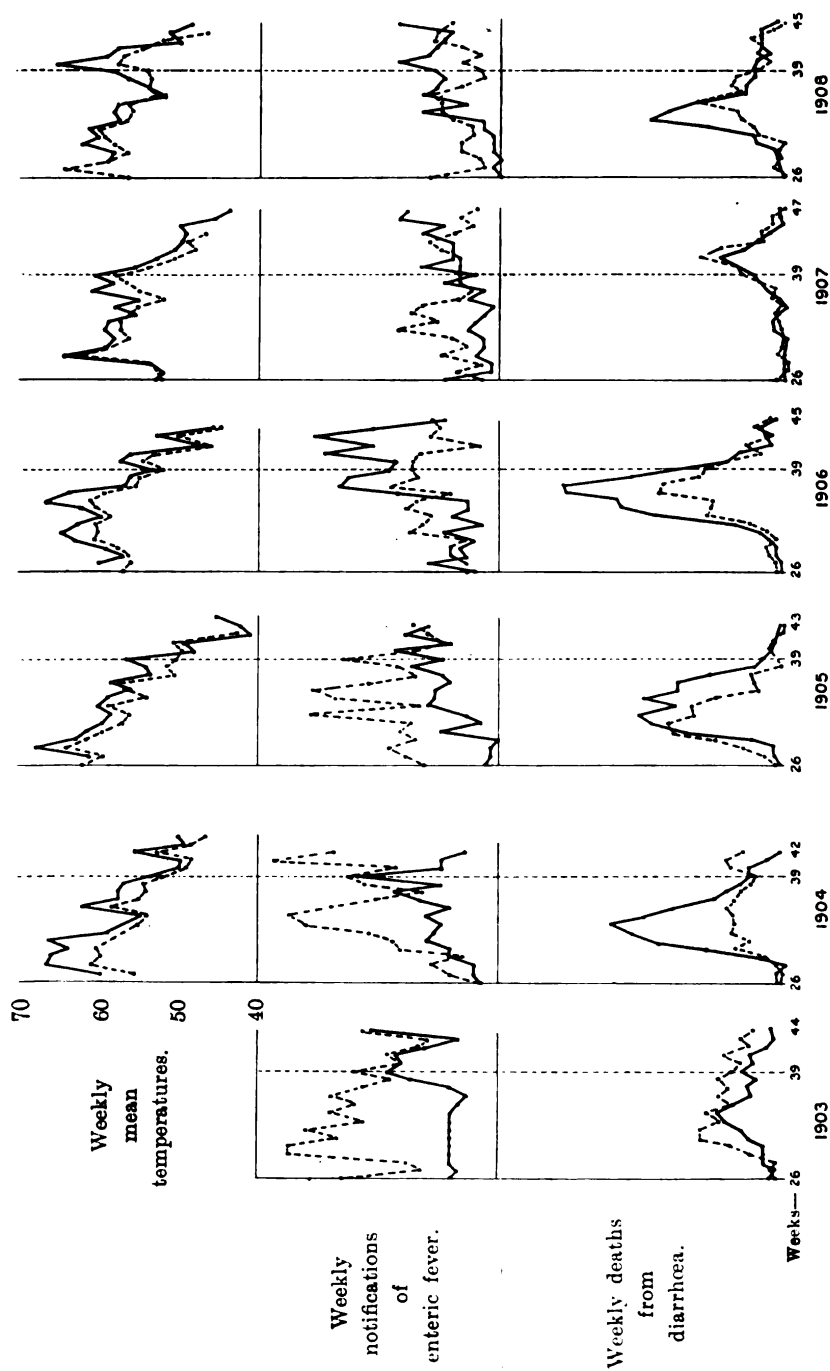


CHART II.—Diarrhoea and enteric fever in Manchester (continuous line) and Belfast (interrupted line) during third and part of fourth quarters in the years 1903-1908; also mean temperatures, 1904-1908.

anything like the same extent. The facts to be faced, therefore, in this connexion are that in these two cities, the behaviour of diarrhœa mortality has been similar, notwithstanding fundamental differences in regard to privy-middens in both, and indeed even in one of them.

In regard to enteric fever, the Belfast weekly notifications have been put back two weeks in the curves, so as to make them more comparable with the Manchester data, since the latter are based on commencement of attack. Also, in order to make the diagrams manageable, the Belfast notifications have been divided by 5 in the years 1897-1902, and by 2 in the subsequent years. The seasonal incidence of enteric fever in Belfast differs considerably from that in Manchester, as I showed in my paper, so that it is not surprising that the close similarity of the diarrhœa curves in the two cities does not appear in the case of enteric fever. On the contrary, the differences in most years are very marked indeed, although clearly there should have been considerable similarity in view of the likeness of the diarrhœa curves, if there is the important relationship between the rise of diarrhœa mortality and the summer rise of enteric fever which is essential to Dr. Niven's hypothesis. There seems to me, however, some difficulty in following this relationship in Manchester. If flies are the cause in common both of the rise of diarrhœa and of the summer rise of enteric fever, there should be some correspondence in each year in the respective rises. Given a high diarrhœal mortality, the inference is, on Dr. Niven's hypothesis, that flies have been particularly numerous, and, on the same hypothesis, that the chances of enteric-fever infection by flies have likewise been greater; so that years of high diarrhœal mortality should be marked, on the whole, by considerable summer rises of enteric fever, and vice versa. But this does not appear to have been always the case in Manchester. Compare, for instance, the diagrams for 1897 and 1898, 1899 and 1900, 1904 and 1906, 1902 and 1908. In Belfast these discrepancies are even more marked. I may instance 1897 and 1898, when enteric fever was at its height in Belfast. Diarrhœa mortality was greater in 1897 than in 1898, but the summer rise of enteric fever was much greater in 1898 than in 1897. Again, in 1905 and 1906, the summer rises of enteric fever do not correspond at all with the respective rises of diarrhœa. Moreover, in Belfast, the summer rise of enteric fever is sometimes prior to that of diarrhœa.

On the whole, the evidence of the Belfast data, while it seems to confirm that of the Manchester data as to possible connexion between flies and diarrhœa, is very conflicting as to the connexion between the summer increase of enteric fever and increase of diarrhœa. It is possible, of course, that in Belfast the exceptional potency of the shellfish factor has obscured the effect of relatively minor influences.

Dr. NIVEN'S reply: I should like briefly to recapitulate my argument as deduced from the relationship of the curves of flies and diarrhoeal cases and deaths, coupled with known physical facts and bacteriological data, and to point out that the effect of flies, in causing a movement upwards of the curve of enteric fever during the height of the season, is of a low order of magnitude, and, in spite of the fact that it might not be in evidence at all, being obscured perhaps by a depression of other sources of infection, did yet, by its peculiar features and tendency to recurrence year after year, point strongly to an influence of flies in carrying enteric fever. When to this is added strong external evidence of enteric fever being thus carried under more favourable conditions, as in the American-Spanish Campaign, in South Africa, and India, the suggestion derived from these curves is greatly strengthened, and in turn supports the conveyance of diarrhoea by flies.

In reply to Dr. Copeman, I would point out that epidemic diarrhoea was defined as a clinical entity, mainly on the ascertained opinion, for each death in doubt, of the medical practitioner certifying, and that by consequence in tracing the relationship of fatal cases to flies, so far as the curves were concerned, no assumption as to the bacteriology of diarrhoea was made at all. It is true this indifference is not absolutely maintained, since, as Dr. Hamer later on points out, bacteriology comes into the argument that any growth on the soil must be of short duration. This argument presupposes that the *causa causans* of diarrhoea is a quickly growing organism. However, there are certain grounds for this opinion, though they may not be conclusive. All bacteriological investigators converge on the field occupied by the *Bacillus entericus*, the *Bacillus coli*, and their congeners. The bacilli causing meat, milk, and cheese outbreaks of diarrhoea have been shown to belong to this group, and, though the evidence is not conclusive, it may yet be said that the probabilities are that the disease is conditioned by a specific bacillus of this group. If so, it will be a quickly growing organism. To this extent the argument is faulty when it is attempted to be shown that we are not to look to anything growing in the soil for an explanation of the wave of summer diarrhoea. But reliance is not placed solely on this line of reasoning. The argument deduced from the distribution of diarrhoea in towns holds, whatever the character of the infecting agency, providing only that it is extraneous to the body. For all parasites it must be true that a well-paved surface must be much less favourable to them than an exposed soil, and hence that growth in the soil cannot be a material factor in the production of diarrhoea. I do not consider that midden-privies play more than a subsidiary part in the production of diarrhoea. That they cannot do so is shown by the experience of Central Manchester, which has no midden-privies and yet has a very high diarrhoea death-rate. But midden-privies add to the influences causing a high diarrhoea death-rate, at all events large, open middens such as prevailed in the outer districts of Manchester. Their absence may not be able to counterbalance the

effects of social depression in Central Manchester, but were they planted now in Central Manchester there can be no doubt that there would be in this part of the city an increase in fatal diarrhœa.

As regards the kind of trap to be used, I admit that the beer-trap prevents the flies from being classified, a matter probably of little moment for this particular purpose, but so, to most people I think, would stick papers. Probably balloon traps have failed in Manchester through failure to bait them skilfully. At the same time a sufficient number have been caught in balloon traps to admit of sample classification.

As regards the continuance of flies from season to season, it is, of course, difficult to assert positively that flies are never continued from pupæ. But it is quite certain now that they not infrequently remain alive all the winter in houses. On the other hand, the circumstances under which they could produce a brood inside a house are very exceptional. Pupæ are easily arrested in their development, and, while flies undoubtedly hibernate in quite a number of places, the materials in which pupæ are found are constantly being removed. It is difficult to imagine where pupæ could remain viable all the winter.

Dr. Copeman takes exception to the dogmatic statement that, in considering the spread of enteric fever, fleas and bugs may be put aside, and asks why it is made. But the reason is given, whether it is or is not considered a satisfactory one; enteric fever visits very sparingly the common lodging-houses and other places of that kind which are the special haunts of fleas and bugs. It is quite true that many cases of fever are covered with flea-bites, but that does not prove that fleas had anything to do with starting their illness. On the contrary, it suggests the question why, if the *Bacillus typhosus* occurs in the blood of patients at an early stage, these do not appear to be infective until later on in the disease, at all events in the great majority of cases. But the want of correspondence between the topographical distribution of enteric and that of bugs and fleas appears to me a strong reason for putting them aside.

The experiences related by Col. Davies and Col. Notter are valuable corroborations of the connexion between flies and enteric fever in tropical countries.

Dr. Hamer blames me for abandoning the cautious philosophical attitude on this question. But important practical issues rest on the view which one takes, and I have not absolutely affirmed the opinion that flies and diarrhœa are synonymous terms. My position is that, to the best of my judgment, a connexion exists of so intimate a character as to call for practical action, and I have endeavoured to lay the facts collected before the Society in such a shape that others may form their judgment upon them. They are by no means such as to permit of the assertion that proof of causal relation has been established. But they do admit of the assertion that no other hypothesis so far advanced will adequately explain the facts, and, on the whole, it seems to me that conveyance by flies does explain the cause of diarrhœa. Possibly this

view will be altered as time goes on. He says the foundations on which the fly theory can rest have still to be laid down. But surely even now it is competent for a clear thinker like Dr. Hamer to tell us what are the foundations on which he would be satisfied to build, so that we may discuss the foundations and arrive at a mutual understanding. I have consistently held that correlation must not be confused with causation. There may, however, be such a closeness of correlation as to justify us in acting on it, as though causation had been more immediately proved, especially if other explanations are shown to be unsatisfactory.

Dr. Hamer challenges the assumption that one attack of diarrhoea protects. Yet it is matter of general experience that the same children do not, as a rule, have second attacks, or in other fashion than the same persons have second attacks of enteric fever, at all events in the same season, which is all that is assumed. As to their power to have a second attack of epidemic diarrhoea in subsequent years, I have not investigated the matter and cannot at the present time give any opinion. At all events, they do not appear to have subsequent fatal attacks, so far as our limited investigations go. Dr. Hamer takes exception to the assumption that the causal organisms of diarrhoea must be of rapid growth. But, as already explained, in objecting to the origin of diarrhoea in the soil, reliance is not placed solely on that line of reasoning, which, it must be admitted, does introduce bacteriology into the question.

Dr. Hamer does not consider that a satisfactory explanation has been given of the fact that the decline in diarrhoea deaths proceeds more rapidly than the decline in the number of flies. No doubt he is quite right in saying that the hypothesis of exhaustion of material is inadequate to explain entirely the whole of the phenomena; and, as a matter of fact, the course of the curves is very similar in the first part of the decline. It is only after half the descent has been accomplished that the curves diverge. It is the later part of the declining curves, the diverging portions, which involve difficulty. It does not follow because the hypothesis of exhaustion is less applicable to years of low diarrhoea incidence that it does not explain much in years of high diarrhoea incidence. It is, no doubt, inadequate as a complete explanation. Hence the fact that flies are immobilized by disease, and not merely by disease, but also by a declining temperature, has been taken into account. It does appear to me that an explanation is thus given of the divergence of the curves. Surely it is not doubted that both facts do restrict the movements of flies very largely, and that such restriction would tend to produce the alterations in the curves which exist. It is, of course, open to a critic to say he wants more proof than the general assertion that these influences exist and may produce the changes observed. Such an accurate fitting of facts I am not in a position to give. But, if the curves fitted accurately, one would be tempted to inquire how it was that these striking facts in the life-history and behaviour of flies were unrepresented.

With regard to Dr. Hamer's observation that the rapid manner in which the cases appear to be produced by flies is at complete variance with the histories of direct infection given in my annual reports for 1904 and 1905, I am at a loss to understand on what he bases his computations. The cases have been picked out which appear to throw light on the latent period in what has been called "direct infection;" and, that there may be no dispute as to the interpretation, these are given under the articles on diarrhœa in 1904, taken from those regarded as affording stronger evidence, being the cases numbered 6, 7, 10, 11, 13, 20, 27, 28, 34, 44, 45, 48, 53, 61, 69, 73, 77, 82, 84, 88, 96, 100, 104, 106, 109, 110; in 1905, Cases 1, 2, 3, 4, 20, 21, 23, 25, 28, 29, 33, 34, 40, 45, 46, 48, 51, 53, 54, 56, 57, 59, 60, 61, 62, 63, 71, 72, 73, 76, 79, 80, 81, 83, 86, 87, 90, 91, 94, 100. Under these numbers there occur, in 1904, twenty-eight cases; in 1905, sixty cases connected with previous attacks. An analysis of the latent periods occurring under these numbers appears to show that there are almost no definite latent periods. In Case 69 (1904) the definite latent period is about a week. In Case 106 it is five days. In each of Cases 51 and 57 (in 1905) it is one day. There are, however, a few cases in which death is succeeded by a subsequent attack. Now we must not assume that infection ceases with death. Owing to handling of clothes, and other causes, we cannot assume infection to cease before the burial of the child, while there is, at this period, special risk of a slight attack being overlooked. There are seven such cases, in which a subsequent attack occurred after a death—viz. (110) in 1904, at an interval of nineteen days; (3) of the year 1905, at an interval of seven days; (54) interval seven days; (60) interval nine days; (62) seven days; (81) eight days; (86) five days. In (3) the second case was the mother, who was attacked seven days after the death of her child. In Case 60 the history is complicated by the circumstance that two grand-parents, who often visited, suffered from diarrhœa prior to the onset of the attack in the child who died, and may have remained infective. In Case 62, also, the father was ill with diarrhœa before the fatal attack began, and may have remained infective. In the great body of the cases, however, exposure to infection was continued right up to the second attack. They may be thus classified. The second attack began while the case was still exposed to infection, but within the following number of days after the onset of the first attack:—

	Within 7 days	From 8 to 14 days	Over 14 days
1904 and 1905	34	20	11

The 34 cases of seven days or under include 7 described as occurring "in a few days," "in a day or two," "directly after." These figures are not incompatible with the curves, if we suppose the fly the infecting agent. If they were, there would not be the slightest justification in taking the above periods as being those of latency. On the contrary, the likelihood is that, the nearer a fatal case to its termination, the more infectious it is; so that, generally,

infection may have occurred near the date of the second attack. In addition to the above there are a few cases in which infection was not continued right up to the attack, the periods of exposure before the attack being, in days: 2 and upwards, 17 to 6, 16 to 1, 26 to 20, 10 to 8, 12 to 4, 10 to 3, 8 to 1, a fortnight to a week, 26 + a few to a few, 6 and upwards, 22 to 5. In all this there is nothing inconsistent with the great majority of the infections being well within a week. It is not necessary, therefore, either to abandon these clinical histories of infection or the average short period of infection required to account for the numerical relation of flies to fatal cases commencing. On the contrary, I would refer to the histories of "direct" infection in 1904 and 1905, as I am confident they would produce on the mind of any impartial person the same impression of direct conveyance of infection which they did on mine.

Dr. Hamer quotes my observation, of a date preceding these inquiries by some years, that diarrhoea shows no signs of being infectious. I had not then realized that there is a necessity for special searching inquiry in the elucidation of this order of facts, as much as in the case of tuberculosis. Surely I do not ask for periods of latency of five or six weeks in the case of enteric fever. An examination of the curves will show that the case rests, such as it is, on the primary wave, which nearly coincides with the curve of fatality from diarrhoea. Indeed, this part of the argument has been left to the impartial examination of the curves by the readers of the paper. As an effort has been made to show why the typhoid summer waves declined in later years, the reader is referred to the text and charts.

Dr. Parsons' interesting observations raise a point of difficulty, and smooth it over very satisfactorily. It unfortunately happened that no fly observations were made in 1907, so that there was a break in continuity. But the year is not needed to show how closely flies and diarrhoea go together. I regret, with Dr. Wheaton, that the figures are incomplete. But the same difficulty meets one all the way through—that correlation is not causation—and would still beset the year 1907, whatever the relations.

Dr. Buchan appears to have hit the weakest point in the argument for the causal connexion of flies with diarrhoea fatality when he points out that at the apex of the curves we should expect the curve of cases commencing to continue after the fly curve has begun to fall. The closeness of correspondence at this point is unfavourable to the connexion. This is true, and I am not prepared, at present, to explain to his satisfaction why there is no such continuance of the curve of cases commencing beyond the curve of flies, as might be anticipated. It may, however, be pointed out that already a large number of infants have been attacked, by hypothesis, as the result of introduction of infection by flies into the household. It is thus becoming increasingly difficult, near the apex of the curves, for flies to raise the number of cases attacked, so that it is quite possible that when the maximum number of flies is reached, although the amount of infective matter has now greatly augmented, the field

of operations of the fly has already become greatly restricted, so much so as to cause any decline in flies to evoke an immediate response in decline of cases. This should be least in evidence in years of low fatality, although it may be pointed out that fatality does not necessarily correspond to incidence. It is, I think, least in evidence in 1905, 1908, and 1909. In 1908 a secondary small rise in flies is followed by a rise in cases in the week following. It is, however, perhaps desirable not to push minor features of the curves, which, both for flies and diarrhœal cases, are admittedly inadequate. It would, perhaps, be unwise to insist on the gradual increase of the fly fungus as a retarding influence at the apex of the curves. Yet I submit that to disregard the facts of the fly's life-history is to court defeat in our inquiries.

While I admit, then, that Dr. Buchan's contention is one which should be submitted, and its force acknowledged, I do not admit that it materially impairs the probability of a connexion created by other features of the curves. One such feature is the slower rate of increase of cases as compared with flies at the commencement of the ascent, and its more rapid rate in the later stages of the ascent. This accords with the result of multiplication of foci of infection. It may be said that what we require is a rate of ascent representing increase of flies multiplied by increase of foci. By no means. It must not be forgotten that the operative flies are gathered round previous foci of infection. Hence the probability of limitation near the apex of the curve. And, in fact, in a year of moderate intensity the numbers already attacked at the height of the diarrhœa season near fatal cases has been shown in some instances to be considerable. For illustrations of this point I must refer to the details of cases in the reports for 1904 and 1905 on the health of Manchester. It is a point, however, which deserves special and accurate study. For reasons already given, I cannot accept Dr. Buchan's observations on the immobilization of flies.

Further, as regards the apex of the curves, I might ask what there is at this point to arrest the multiplication of flies, now in enormous numbers. The meteorological data scarcely seem adequate to explain this arrest. A full answer to this conundrum might throw light on the arrest of cases.

Dr. Buchan's concise and forcible exposition of the difficulty relative to the apex of the curves is, it is proper to remark, in a measure equivalent to Dr. Hamer's criticism, when he in effect says that one cannot have it both ways, that flies are becoming immobilized during the descent of the fly curve for purposes of diarrhœa, but are mobile and active for the purposes of enteric fever. That is quite true, but I do not claim a large amount of immobilization, though an increasing one in the early part of the descent. What has been put forward is that the primary part of the enteric ascent manifest in the majority of years is probably decidedly smaller than it should be, owing to the increasing numbers of enteric fever being mixed up with diarrhœa. It is to this earlier portion that I would direct special attention in connexion with flies. The

secondary rise or rises, antecedent to the thirty-eighth week, are dependent probably on these primary rises, which nearly coincide with the upper part of the fly curve, allowing for the ordinary incubation periods of enteric fever. They arise from them partly by direct infection from the overlooked cases, which have been shown to be not infrequently confused with diarrhoea, and to be specially concerned in causing increases of enteric fever, and partly they are due probably to the movement of flies in the early part of the descending curve, conveying infection from the recognized and unrecognized cases concerned in the primary rise. There is no question of a delay of five or six weeks, and no need to suppose either for diarrhoea or enteric fever that, at this period, there has occurred extensive immobilization of flies, although, as a matter of fact, Mr. Gordon Hewitt states that the fly fungus is already extending at this period.

Dr. Darra Mair's communication is an exceedingly interesting one. So far as criticism of the paper is concerned, however, I am only concerned with his remarks on the course of the enteric-fever curves in Belfast. It does not follow because the summer course of enteric fever is different in Belfast and in Manchester that we must reject or disallow the impression which the Manchester curves make. As Dr. Darra Mair observes himself, perhaps the effects of shellfish in Belfast obscured those with which we were dealing in Manchester. There is, I believe, no close season in Belfast as there is for English mussels, and in all probability the influence of mussels there would be markedly in evidence during the early fly season, when here it was almost absent. Not only so, but I could not accept the antedating of weekly notifications by a fortnight as in any way representing commencement of attack, although on this point it is possible that Dr. Darra Mair may have some explanation to give. If, however, I understand him correctly, his curves of commencing cases would not be comparable with those for Manchester, as the determination of the dates of onset would probably materially alter the shape of the curves. With regard to his observation that years of high diarrhoeal mortality should be marked, on the whole, by considerable summer rises of enteric fever, and vice versa, this is certainly a valid one. "*This does not appear,*" he says, "*to have been always the case in Manchester. Compare, for example, 1897 and 1898, 1899 and 1900, 1904 and 1906, 1902 and 1908.*" But an examination of the curves for 1897 and 1898 scarcely supports the above statement. Not only is the shape of the diarrhoea curve, and therefore of the fly curve, of 1898 more favourable to the production of a high enteric incidence, but the aggregate amount of fly influence as measured in the same way is greater. The area of the diarrhoea rise in 1898 to a corresponding part of the rise in 1897 is as 173 to 84, and the total areas of the diarrhoea rises are as 192 to 120. A comparison of 1899 and 1900 does show a somewhat greater rise in the later part of the 1900 enteric curve, which, if it was necessary to strain the argument, might reasonably be ascribed to the greater continuance of the diarrhoea

maximum—with, by hypothesis, a similar distribution of flies. It is, however, only after the mussel season is reached that the enteric curve of 1900 goes in advance of the earlier year. Up to that point such differences as exist are of little moment. Comparing 1904 and 1906, the rise in 1904 is earlier than in the latter year, a fact which may well be associated with the greater number of cases in the earlier part of the year in 1904. The total effect produced, however, and the magnitude of the producing causes, as measured by the diarrhœa curve, are much alike up to the thirty-eighth week. A comparison of 1902 and 1908 shows that in the former year the "primary" rise of enteric, as compared with the diarrhœal rise, is large as measured against that in 1908. There are, however, two facts to be considered in the comparison. One is that in 1902 both diarrhœa and enteric fever are late, so that the enteric rise has the assistance of the mussel season, the effects of which begin to be acutely felt in the thirty-eighth week. The 1902 primary rise begins in the thirty-eighth week. Another fact is that the primary rise of enteric fever is superadded on a fairly considerable previous incidence of enteric fever in 1902, while that of 1908 springs from nearly zero, the curve having previously nearly touched the base line for seven weeks. The fact is that the impression derived from these curves is the result of careful study, and is difficult to convey in words. The rise of enteric fever connected with flies is a slight one, and the phenomenon is variable in amount and character, as we should expect; but a decided impression remains from their study that, whatever be the causes operating to produce the diarrhœa uprush, they are also concerned in producing the primary rise in the enteric curve. Possibly my attitude has been too dogmatic in this question. I admit the force of some of the difficulties and criticisms put forward. But my impression as to the important rôle played by the house-fly remains unaltered.

I beg to be allowed to acknowledge my obligations to all who have assisted me in compiling the data or in making observations, more especially to Mr. Dunks, of the Public Health Office, to Dr. Duncan Forbes, Dr. B. K. Goldsmith, and Dr. J. R. Hutchinson.

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

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Laryngological Section.

November 5, 1909.

Dr. J. DUNDAS GRANT, President of the Section, in the Chair.

Chronic Glanders in a Man aged 24.

By G. SECCOMBE HETT, F.R.C.S.

THE following specimens were shown: (a) A coloured drawing of the pharynx, showing the loss of the soft palate and ulceration and scarring of the fauces and pharynx; (b) The lungs, liver, spleen, and kidneys—the lungs and spleen present glanders nodules, the liver and kidneys amyloid changes; (c) *Bacillus mallei* cultures on potato made from pus taken from the subcutaneous abscesses; (d) A guinea-pig which had been injected intraperitoneally from the cultures, showing swelling of testicles and thickened and adherent tunica vaginalis.

Mr. Seccombe Hett said that the case had been shown before at the Section, when the consensus of opinion had been that it was a case of tertiary syphilis. The only apparent lesion until a short time before death (when subcutaneous abscesses had appeared on the face and neck) was ulceration within the mouth. The pillars of the fauces, tonsils, and soft palate were destroyed. The hard palate perforated late in the course of the disease. Every form of mercurial and iodide treatment had been employed, but had had no effect. A full report of the case had appeared in the *Lancet*.¹

DISCUSSION.

Dr. W. HILL asked whether the lesion in the throat at all simulated tuberculosis. He recently had a case which was suspected of being glanders, but several guinea-pig tests did not succeed, and the organism had not been found when the patient died within six weeks of admission to hospital. Though no tubercle bacilli during life were found, at the post-mortem the lesion resembled

¹ 1909, ii, p. 1201.

that of galloping consumption. There was a mouse-bitten form of superficial ulceration in the oesophagus, trachea, and larynx, and there were tubercle-like lesions in the lungs and elsewhere; on the other hand, the lesions on the palate and in the skin were strongly suggestive of glanders.

Dr. FITZGERALD POWELL said the Section ought to feel very grateful to Mr. Hett for having brought the case forward. It was interesting not only because the clinical appearance resembled that of tertiary syphilis, but because it seemed to show that there were more cases of chronic glanders than had been previously recognized. He asked whether the mallein test had been used and what treatment was adopted in the later stages of the case.

Mr. HERBERT TILLEY said the case was seen by Dr. Lieven, of Aix-la-Chapelle, when he was visiting Mr. Tilley's clinique, and Dr. Lieven was impressed with the idea that it was tertiary syphilis, particularly as the ulcerated margin resembled that disease, and as there was a large stellate scar on the posterior surface of the pharynx. Mr. Tilley suggested that Dr. Lieven should try Wassermann's reaction in the case. He did so, but the result was negative; yet, in spite of that, Dr. Lieven wrote to say he still thought the lesion was syphilitic: but Wasserman's reaction in that case proved to be right.

Mr. HETT, in reply, said that the appearance was like that of tertiary syphilis. The only thing which looked more like tubercle was that the lesion slowly spread, leaving a trail of scar-tissue behind it. This was especially well seen in the position of the faucial pillars. After the organism was obtained a vaccine was made and the patient injected with it; that was only shortly before he died. There had been great difficulty in getting the organism, and this was only done when abscesses appeared and cultures were made from the pus. The diagnosis could be made, on the history, the temperature chart, and the progressive nature of the lesion despite treatment, even when the *Bacillus mallei* had not been identified.

Cases of Neoplasm of the Tonsil.

By G. SECCOMBE HETT, F.R.C.S.

Case I.—A boy, aged 5, was admitted to the Throat Hospital, Golden Square, with an enlarged right tonsil and a mass of enlarged glands on the right side of the neck. There was a thirteen weeks' history of enlargement of the tonsil. The tonsil was removed together with the palatoglossus, palatopharyngeus, and portion of the superior constrictor muscle. On September 5 the glands were removed from the neck, together with portions of the parotid and the sterno-mastoid. On September 15 a mass was noticed behind the posterior pillar, and this

was removed. On October 12 growth was found on the soft palate and was removed. The growth of the tonsil was the size of two walnuts and was very soft. The mass in the neck was the size of a small orange; growth on section was a sarcoma. The patient and macroscopic and microscopic specimens were shown.

Case II.—Patient, a man aged 46, was admitted to the Throat Hospital, Golden Square, on May 6, 1909. The right tonsil was enlarged. He had a history of a quinsy five weeks before. No enlarged glands in the neck. Both tonsils were enucleated; no recurrence. The growth on section proved to be sarcomatous. The patient and macroscopic and microscopic specimens were shown.

Case III.—Specimen of a neoplasm of the pharyngeal tonsil, with section. Patient, a youth aged 17, died three months after operation.

Case IV.—Specimen of neoplasm of tonsil in a man aged 65, with section. Both tonsils were involved; patient died three weeks after removal of one tonsil for the relief of respiration, which was becoming obstructed.

An Attachment to von Brünings' Instrument for Facilitating Manipulations under Direct Laryngoscopy.

By G. SECCOMBE HETT, F.R.C.S.

THE instrument was designed by von Brünings and has two advantages: (1) That it enables a view to be obtained in cases where the neck is so fat or rigid as to make this difficult by the direct method; (2) That the larynx is pressed backwards, thus obviating the necessity of keeping up forward traction on the handle of von Brünings' instrument; also, by means of a catch, the whole apparatus is fixed so that both hands are free for manipulative work. The instrument was demonstrated upon a patient.

Perforation of Nasal Septum from Salt (NaCl) Dust.

By DAN MCKENZIE, M.D.

A WOMAN, aged 20, noticed dryness and crust formation in nose six months before coming to hospital. No epistaxis. Four months ago boric-acid powder put into one nostril returned by the other. Pain at

times rather severe. Patient is a packer of table-salt of the old-fashioned kind. In dry and warm weather the salt blows about in a fine dust. She first went to work nine months ago, and complained of her nose three months later. The perforation is extensive, involving the greater portion of the cartilaginous septum. Sections cut from the marginal tissues show typical "giant-cell systems."

The warehouse in which the patient works has been visited by the exhibitor. The work-room is large, airy, and well ventilated. There are eight workpeople—seven female and one male—in close contact with salt clouds, as the flouriness of their hair and clothing abundantly testified. Of the seven females (including the patient), four showed perforations, all small, with the exception of the patient. In one case the perforation lay well beyond reach of the finger. The male, a boy, was a grinder of salt and worked continually in an atmosphere foggy with salt-dust, but, although he had been at this work for two years, there was no perforation of his septum. In other respects the employées seemed unusually healthy. None showed any signs of nasal suppuration.

A specimen showing tissue from margin of septal perforation (prepared by Dr. Wyatt Wingrave) was shown.

DISCUSSION.

The PRESIDENT (Dr. Dundas Grant) said it was curious that there should be giant-cell systems; but, as Dr. McKenzie had gone to the source, there was no doubt as to the ætiology of the ulceration, which came under the heading of trade perforations, as described by Morell Mackenzie in his book.

Mr. DE SANTI said the perforation might possibly, if it were not for the history of other similar cases, be taken to be syphilitic. Possibly Dr. Legge, who was present, might have seen such cases before in his inspections of factory workers.

Dr. LEGGE said he found perforation of the septum common where bichromate of potassium and sodium were manufactured. Some time ago he examined 176 men employed in the manufacture of bichromates in Scotland, and 126 of them had complete perforation of the septum, while thirty others had ulceration which would inevitably lead to perforation, and in their case the perforation was even more extensive than in the case just seen. It extended upwards to the junction of the cartilage with the ethmoid, backwards to the vomer; but the anterior and inferior margins of the cartilage were unaffected, so that in only one or two was there any falling in of the nose. He had found similar conditions in workers engaged in the manufacture of sheep-dip, composed of white arsenic and caustic soda, and very occasionally in the grinding

of metal. Nothing so far tried in bichromate works had been successful in preventing the perforations, though the workers were medically examined every month.

Dr. H. J. DAVIS reminded the Section that in 1905¹ he showed a case—a boy, aged 17—with a similar perforation which was traumatic. The boy worked in some flour-mills, and was constantly in a dusty atmosphere. Some of the members at the meeting seemed to think it very unlikely that a dusty atmosphere could cause such a condition, but he was sure the case was a result of that. He had seen other cases like it, and he saw no reason to doubt that the present case was one coming under the same category.

Mr. CLAYTON FOX said that Kyle quoted many cases showing the effects of irritants in producing perforations—arsenic, cement, flour, and dust of many kinds—so he did not think there should be any difficulty about agreeing with the fact that the condition was brought about by an irritant such as salt. The only strange point was the presence of giant-cell systems. Possibly the resisting power of the septum had been lowered and there had been an invasion of tubercle also.

Mr. ROSE said the presence of giant cells was no proof of the existence of tubercle. Any foreign body, such as a piece of silk, might cause giant cells in granulation tissue.

The PRESIDENT asked whether in the other cases examined there was any evidence of there having been a deflection of the septum. In the present case there evidently was such, and the cavity had been a receptacle for the inhaled dust. He believed Morell Mackenzie pointed out in the cases he described, that some mechanical imperfection added to the presence of angular fragments, sufficed to cause perforation, but that neither cause alone could produce it.

Dr. MCKENZIE, in reply, said he could not find in the literature any other case of septal perforation attributed to salt. He did not think that salt acted upon the septum as a neutral substance in the way that flour and cement did—forming dry crusts or scales which led to nose-picking. His idea was that the salt deposited on the septum destroyed the tissues by its hygroscopic action. The possibility of the perforation in this case being due to tertiary syphilis was negatived by its situation, by the fact that its edges healed without treatment when the patient was removed from the salt-packing, and also by the fact that the other workers in the same room were suffering from perforation. He agreed with the President regarding the influence of the deflection of the septum, for in this case the perforation was larger than in the other workpeople, and in them he had not observed any marked deflection. He had inserted the qualification “old-fashioned” salt because he understood that the newer varieties of table-salt on the market contained other ingredients besides sodium chloride, whereas the salt used in the warehouse he had visited was unmixed.

¹ *Proc. Laryngol. Soc. Lond.*, 1905, xii, pp. 92-33.

Tuberculosis of all the Left Vocal Cord and Inter-arytænoid Space, in a Lady aged 46, completely healed by Two Months' Silence and Sanatorium Treatment.

By STCLAIR THOMSON, M.D.

THIS lady reports that she had some consolidation of the right apex in 1907. In March, 1908, tuberculous disease of the right apex was again diagnosed. In May last she noticed increasing hoarseness, and was sent to me by Dr. Stuart, of Camberley. When examined on July 3, 1909, the voice was hoarse, toneless, and the throat uncomfortable. The left vocal cord was replaced by a red, fleshy, and abraded infiltration. There was infiltration of the inter-arytænoid region, with much catarrh. The condition was shown in the accompanying sketch. There was some dullness and tubular breathing at the right apex. No moist sounds, no temperature. The weight was 7 st. 11 lb. The patient entered King Edward VII Sanatorium, under Dr. Noel Bardswell, on July 17, and remained silent for two months. No tubercle bacilli were found, but she reacted positively to von Pirquet's test. When examined on October 6 she had gained 9 lb. in weight; the voice was clear and strong. There was no trace of tubercle in the larynx, the left vocal cord being again white and smooth and the inter-arytænoid region absolutely normal. The only noticeable point in her larynx was the large opening into the sinus of Morgagni. Family history is negative. The interesting point is the rapid recovery in a patient of this age, when general symptoms are slight, without other local measures than silence.

Tuberculosis of both Vocal Processes, in a Medical Man aged 41, cicatrized with Seven Applications of the Galvano-cautery.

By STCLAIR THOMSON, M.D.

IN 1902 this gentleman entered a sanatorium with early mischief at the right apex and plentiful tubercle bacilli in clumps. They rapidly diminished and disappeared in three months, and he was discharged after eight months as an arrested case. After an absence of six months

he returned to work in the sanatorium as one of the medical staff, and remained there for three years. In July, 1907, he took up practice in a large provincial town. All went well until November, 1907, when he had a slight rise of temperature and hoarseness. This never cleared up. He kept at work until September, 1908, when he first came under observation. There was then recrudescence at the right apex with a few tubercle bacilli. Temperature did not pass 99° F. He could walk seven miles without fatigue; he had lost a little in weight, being 13 st. 1½ lb., instead of the 14 st. at his leaving the sanatorium.

The voice was rough and harsh, but not painful. Over the whole right vocal process was a nodular ulcerating surface, spreading up to the front of the arytæmoid. Over the left vocal process was a similar condition, but the ulcer was deeper as the projection on the right side fitted into it. The patient had seven applications of the galvano-cautery, which were made between October 8, 1908, and May 20, 1909. During this period he has carried on a practice single-handed, and on September 28 he wrote as follows: "My voice is wonderfully good, a little roughish perhaps. General health good. To-day I have cycled twenty-one miles, and feel equal to the same distance to-night if necessary."

Tuberculosis developing in the larynx after the process has been arrested in the lungs does not generally assume an acute form, but it is apt to be extremely tedious, and the above case shows how a cure may be effected, even without rest to the larynx, and while the patient is fulfilling arduous duties.

Extensive Tuberculosis of the Epiglottis, left Ary-epiglottic Fold, and Left Inter-arytæmoid Space, in a Gentleman aged 47, completely healed by the Galvano-cautery and Sanatorium Treatment.

By STCLAIR THOMSON, M.D.

THIS gentleman first complained of sore throat in the summer of 1908. He brought up a teaspoonful of blood, which led to examination of sputum, with positive result. In November, 1908, he was taken by his medical adviser (Dr. Blatherwick) to consult Dr. Newton Pitt, who found tuberculosis of larynx and lungs.

When admitted to Pinewood Sanatorium in November, 1908, there were rhonchi over both apices masking the finer sounds. The sputum contained tubercle bacilli, and there was infiltration of the epiglottis, inter-arytænoid region, and left ary-epiglottic fold. The chest improved, but the laryngeal condition became more marked, and, as absolute silence failed to effect an improvement, he was referred to me by Dr. Herbert J. Phillips in February, 1909. It was then seen that the entire epiglottis was thickened, red, velvety, and overhanging the glottis. All its outlines and contours were lost. When the patient phonated, it was seen that the laryngeal surface of the epiglottis was ulcerated. The vocal cords, the right arytænoid, and the right ventricular band were intact, but there were rolls of indolent infiltration of the left ary-epiglottic fold and pale pink deposit in the left inter-arytænoid space. The patient was treated with the galvano-cautery, and between February 8 and September 30 he received seven treatments. Improvement was marked from the first, although the sputum continued to show numerous bacilli, and the general progress was interrupted by an attack of appendicitis (? tuberculous) and a temperature of 105° F. in June.

It will be seen that the larynx is now soundly healed, although nearly all the epiglottis has been destroyed. The director of the sanatorium observed that, in spite of a fair amount of active mischief in the chest, the temperature began to improve with the improvement in the larynx.

DISCUSSION.

Mr. HERBERT TILLEY asked to what kind of case Dr. Thomson would apply the galvano-cautery. Would he apply it to infiltrations only, or to the superficial as well as to the extensively ulcerated case? It had happened to him (Mr. Tilley) twice that day to apply the galvano-cautery to patients with tuberculous laryngitis. To one of them he had now applied it four times. It was by the direct method, with a long galvano-cautery point. The case to which he had applied it four times was an infiltration, and the patient for whom one puncture had been made had superficial laryngeal ulceration, which he cauterized under the impression that it would be the quickest way of getting rid of it. Twelve months ago he applied the galvano-puncture (in a patient from Midhurst) to a left ventricular band, which, owing to infiltration, completely hid the corresponding vocal cord. She had the ordinary symptoms of tuberculosis of the lung, and bacilli were present in the sputum. The laryngeal trouble had so completely cleared up that he could scarcely tell from the appearance alone which had been the affected side. The cases which seemed to

do better by the treatment were the infiltrated cases. No doubt Dr. Thomson had an excellent reason for cauterizing the third case, but he would like to know why he preferred the galvano-cautery to the amputation of the free and diseased portion of the epiglottis. The speaker had successfully adopted the latter measure when the free portion of the epiglottis was affected by tubercle. Amputation shortened the treatment materially—there was no need for constant punctures.

Dr. WATSON WILLIAMS asked for a little more detailed information as to what was done to the epiglottis by puncturing, particularly whether they were deep, fine punctures or superficial, and whether there was a local reaction in that case; and did Dr. Thomson attribute the excellent result mainly to the local reaction rather than to the direct action of the cautery on the tuberculous lesion? All members would join in congratulating Dr. Thomson on his excellent results, especially as he was certainly one of the very first English authorities to emphasize the value of pure rest for tuberculous cases. He was very glad the exhibitor also resorted to other methods than simple rest, which in many cases were very useful accessories to the "rest" treatment.

Dr. SCANES SPICER congratulated Dr. StClair Thomson on the success of his measures as far as the larynx was concerned. He noticed, with pleasure, that he had used the words "healed" and "cicatrized" in all three cases, instead of, as had been often seen elsewhere, the word "cured." The left cord in the woman was still decidedly thick, and in Case 3 the frænum of the epiglottis was still in a suspicious state of swelling and redness; but the cases were immensely better, and a credit to Dr. Thomson's laryngeal technique. What he wished, however, to specially point out on the present occasion was that the three tuberculous cases shown were "abdominal" or "belly" breathers—i.e., inspiration was effected by the descent of the diaphragm and viscera *en masse*, the belly wall being translated forwards. The mechanical effect of this on the circulation (as compared with costal inspiration combined with fullest vertebral extension—i.e., "back" breathing) was, relatively, to hamper efficiency in the pulmonary circulation, to lessen the percentage oxygenation and decarbonization of the blood in the lungs, and in the systemic circulation area to impair the vitality and resistance of the tissues throughout the body, including the lung substance. The sooner this great truth was recognized and put into practice all round, the better for the science and art of health, the public, and the practitioner. It was not a little suggestive that one of the co-authors of a leading modern monograph¹ on the vocal organs, who strenuously championed, taught professionally, and personally practised "abdominal" breathing, died of lung tuberculosis. The speaker could multiply instances indefinitely from his own experience of the ill results of exclusive habitual "belly breathing," and the extraordinary improvement which ensued

¹ Incidentally, the other co-author, who widely advocated and practised "abdominal" breathing, died of cancer of the stomach. Compare speaker's working hypothesis on these problems. *Brit. Med. Journ.*, 1909, ii, p. 673, 1149.

when the vertebral column and ribs in inspiration, and the belly wall in expiration, were used to full mechanical advantage. This principle was second to none in dealing with the tuberculosis problem in man, both in prevention and treatment, local and general.

Dr. DONELAN said that last May he showed a bad epiglottis case, and it was suggested that an alternative treatment would be removal of the epiglottis or the galvano-cautery. He applied the latter fifteen times, and the patient made fair progress during that time, but much greater when the silence treatment was strictly carried out in addition. He would bring the case to the next meeting.

The PRESIDENT said he could completely support what had been said by Dr. Thomson, Mr. Tilley, and others as to the value of galvano-cautery puncture. He had advocated it at the Section, and was strongly of opinion that the infiltrative were the forms which were benefited by it. He had asked the sister in charge of his department at Brompton which cases did best, and her reply was those for which he used the galvano-cautery. No doubt they were the cases selected as appropriate for that. Infiltrations, especially when they were more or less circumscribed, were benefited, and the surprising thing was that the patients got an extraordinary relief from pain, even when the puncture was made close to an ulcer which had been painful before. He had sought for rises of temperature after that somewhat active treatment, but none had been present. He admitted he was not now so keen about amputation of the epiglottis as formerly, and for two reasons: in one or two cases where he removed the epiglottis, perhaps somewhat thoroughly, the framework of the larynx had been already so infiltrated that it could not close in so as to prevent the entrance of fluids during drinking. No doubt if the removal of the epiglottis had not been effected at the time the difficulty would have come on just the same. The instant relief from pain was, however, most striking. The other reason for which he had practically given it up was that the galvano-cautery produced such excellent results. The sclerosis produced in that way was remarkable in its benefit. No one would dream of failing to utilize a single factor, and he always enjoined silence, as far as it could be obtained, as well as the galvano-cautery.

Dr. STCLAIR THOMSON, in reply, said that in regard to the selection of the cases most suitable, it would be difficult to describe his choice in a few words, but he would be inclined to try that treatment in all cases where the general conditions were favourable, and where the laryngeal disease did not show any approach to the arytaenoid joint, and where the march of the case was not of that rapid nature which it was difficult to describe in words. One knew in such cases as soon as one looked into the larynx that it was no good doing anything at all. He had applied it in cases of pseudo-œdema which formerly he had been afraid of, and in a few of those he had been successful. His cases had been carefully selected, and he had never had one drawback; every case in which he did the treatment had been completely cicatrized. As Mr. Tilley said, infiltrations did best of all, and he began on them particularly in the

epiglottis and ary-epiglottic folds and the ventricular bands. He also did it for ulcerated cords and subglottic ulcers if the general appearance was favourable, and if it was not a rapidly-advancing case and not invading the ary-tænoid joint. There had been numerous bad results from amputation of the epiglottis. He had heard of unpublished cases in which amputation of the epiglottis had set up acute miliary tuberculosis, and that was what one would expect from pathological teaching. The object of the galvano-cautery was not to burn away the disease ; but it acted by stimulating fibrosis, which, as Dr. Horne had said, was Nature's method. Amputation of the epiglottis did not do that at all, but, rather, laid waste all the lymphatic channels and opened the whole area up to pyogenic infection. As the epiglottis in this patient was so vastly infiltrated and pendulous he suggested amputation, but the patient declined, saying he would rather have the cautery. He only had seven visits ; the treatment was not painful, and there was no risk about it. At first he used to keep patients in town for the first day ; but now they went back to the sanatorium by the afternoon train on the same day, and he had no fear of reaction in properly selected cases. In answer to Dr. Spicer, probably there were still tuberculous deposits in one case ; but there came a stage in those cases when, if the treatment was persisted in, they did not seem to progress, whereas they seemed to get well if left alone. He was not sure whether it was tuberculous disease in the fold which had been referred to, but when such cases arrived at the stage of fibrosis the sanatorium treatment kept up the general health, and in a month or two probably the disease would have gone.

Papilloma of the Larynx in a Boy aged $6\frac{1}{2}$, of four years' duration, cured by Tracheotomy and repeated operations by direct Laryngoscopy.

By STCLAIR THOMSON, M.D.

THIS boy was so blocked up with laryngeal papilloma that a tracheotomy had been performed on him before he was admitted to King's College Hospital in 1906, at the age of $3\frac{1}{2}$. There was hardly any air-way through the glottis, so that the patient had learnt to speak fluently with the pharyngeal voice. Numerous operations were performed on him during the years 1906, 1907, 1908, and 1909. There is no correct record of how often he was put under chloroform, but there are notes of a general anæsthetic being given and of the papillomata being removed by direct laryngoscopy on sixteen occasions. Arsenic was also administered. The growths continued to recur until this year. Since Christmas he has spoken with the glottis, and allowed the tracheotomy

cannula to be corked for part of the day. He still had to wear it when he was discharged on April 4. He was readmitted to the medical wards with measles from May 18 to 29. When taken back to the throat ward on June 30 it was found that his larynx was quite free, and that he could breathe perfectly freely with the tracheotomy tube blocked. The cannula was therefore abandoned on July 2. He now has a very good voice, and if gently handled can show his larynx by ordinary laryngoscopy. There is a small fistula over the tracheotomy wound, and it is proposed to close this by a plastic operation.

The case is interesting as showing the persistent recurrence of papillomata in spite of frequent and complete removal. It also shows the natural tendency for laryngeal papillomata to disappear about the age of 6. Is it possible that the attack of measles had anything to do with their final disappearance? The case is also shown to illustrate the advantage and harmlessness of wearing a tracheotomy tube, and how a patient may be cured with a good voice. For many years protests have been made against the useless and crippling employment of laryngo-fissure in these cases. It is now deplorable to note that at the last meeting of the Belgian Society of Laryngology, it is proposed to submit these poor children to laryngo-tracheostomy.

DISCUSSION.

Dr. PATERSON said he had had three or four such cases where the result had turned out admirably. One was exactly similar to the one Dr. Thomson showed that day, and he (Dr. Paterson) had recently done a plastic operation for the fistula which had remained after the tube. He inspected the larynges of two of them by the direct method, and was surprised at the width of the air-passages. He was in complete sympathy with Dr. Thomson's protest against subjecting such children to laryngo-tracheostomy. Repeated removal of the growths by the direct method and the wearing of a tracheotomy tube, if necessary, ought instantly to be given a prolonged trial.

Dr. STCLAIR THOMSON, in reply, reminded Dr. Paterson that he had seen the patient and helped him once with the removal; it was an obstinate case. He did not claim that the cure had been altogether effected by the removal; the papillomata returned rapidly, but as the boy grew older the recurrences were less acute.

Woman, aged 56, after Laryngo-fissure for Subglottic Enchondroma.

By STCLAIR THOMSON, M.D.

THIS patient was shown by Mr. Stanley Green and Dr. Lambert Lack at a meeting of the Section in April, 1908.¹ She then had a smooth, mound-like growth springing from the posterior wall of the larynx below the vocal cords. The exhibitors were inclined to consider it a tuberculous tumour, and in view of the facts that there was evidence of tuberculous disease of the lungs, as shown by X-ray examination and reaction to Calmette's test, several members were opposed to active operative interference. Dr. Watson Williams and Dr. Grant diagnosed the growth as cartilaginous. The patient afterwards passed under the care of Sir Watson Cheyne (by whose kind permission the case is now exhibited), and he removed the tumour by laryngo-fissure. Microscopic examination showed it to be an enchondroma.

DISCUSSION.

Mr. STANLEY GREEN asked whether the patient had derived any benefit from the operation. He had watched her carefully for two and a half years, and he could not find that her voice was improved, or that she had any larger opening to breathe through. Moreover, she had now an extensive keloid of the neck. He did not think it was worth while for the patient to have gone through the operation.

Dr. STCLAIR THOMSON replied that he was not responsible for the operation. She was uncomfortable that day because of the London fog, which had lined the larynx with "blacks." She previously had a good deal of stridor, which the operation had relieved.

Cases of Extra-laryngeal Inoperable Carcinoma, shown to illustrate the Beneficial Effects of Operation on the Thyroid Gland.

By W. STUART-LOW, F.R.C.S.

Case I.—The man was a porter in the General Post Office, who came to the clinic at the Central London Throat, Nose, and Ear Hospital complaining of hoarseness and difficulty of swallowing of two months'

¹ *Proceedings*, 1908, ii (Laryngol. Sec., p. 90).

duration. He gave a history of repeated attacks of influenza, but not of syphilis. A large, grey, mushroom-like mass was seen covering over and projecting into the larynx. About one-third of this mass was removed with forceps; this gave him considerable relief. Dr. Wyatt Wingrave reported that it was certainly a very rapidly growing epithelioma—"one of the most virulent that he had ever seen." In the discussion which followed at the sectional meeting Mr. H. Tilley said that he thought the growth was certainly of a malignant nature and far too extensive to be operated upon. He also emphasized the presence of many enlarged glands in the neck. The patient was shown to the Society on May 7, 1909. On May 10, 1909, under local anæsthesia (1 per cent. cocaine) a collar incision was made over the thyroid, the isthmus was divided, the left lobe isolated, and all the vessels proceeding to and from this ligatured. Suddenly the larynx became obstructed, probably from the large growth hanging over its entrance having become fixed in the passage. The patient very rapidly became cyanosed and respiration ceased. It was urgently necessary to perform tracheotomy, when recovery quickly took place. This accident proved the wisdom of having used local anæsthesia as against general anæsthesia in the particular case. It was now found impossible to remove the thyroid without greatly prolonging the operation, and it was thought best to tie the superior thyroid of the right lobe, and trust to this and efficient ligation of the left lobe to minimize the thyroid function. The large wound was partially closed and firmly packed with gauze. This patient made an excellent and uninterrupted recovery, leaving the hospital in a fortnight: the left lobe of the thyroid, all the vessels of which had been ligatured, sloughed and came away in the dressings with the ligatures. It is now five months since the operation was undertaken, and the case has been watched very attentively. Before this operation the patient was rapidly getting worse and losing weight, but since then he has been putting on weight, having gained 1 st. 1 lb. He is now swallowing better and the growth in the larynx and pharynx has diminished very much in size. There has been no pain, and the patient is much stronger and expresses himself as feeling better in every way since being operated upon. This is the first case in which ligation, as distinguished from excision, has been practised and trusted to for the partial ablation of the thyroid. Judging from the results in this instance, the plan would certainly seem to answer well. Ligation is a more rapid process than excision, and the thyroid in these cancerous subjects is often enlarged, very vascular, and very adherent

to surrounding structures, therefore often most tedious and troublesome to isolate.

Case II.—The case was that of a man, aged 66, with epithelioma of the soft palate and some secondary glands on both sides of the neck. On July 10 hemithyroidectomy was performed under local anæsthesia. He is still under observation and has gained 6 lb. in weight. His general condition is good and the glands have become smaller and softer. He is attending regularly as an out-patient.

Case III.—The third case was that of a man, a painter by trade, aged 58, who came to the clinic at the Central London Throat, Nose, and Ear Hospital on May 22, 1908, complaining of pain in the tongue and neck and of a swelling on the neck of six weeks' duration. There was an ulcer of the size of a shilling on the right side of the tongue. It was excavated and indurated, and had the characteristic stony hardness around and towards the base of the tongue. The induration extended on to the palate and under the sterno-mastoid near the angle of the jaw. No doubt could be entertained as to the primary ulceration being epitheliomatous, or that the enlarged glands were a secondary extension from the tongue. The pathological report supported the clinical diagnosis, and, it being considered futile to try to eradicate the growth, thyroidectomy was decided upon. Complete hemithyroidectomy was performed on June 3, 1909, the left half of the thyroid, including half of the isthmus, being removed; it was found very vascular and adherent to the surrounding structures. The patient was in the hospital for a week after the operation, and during this time suffered no pain in the tongue or neck. For some time before the operation the pain in the neck, shooting from the tongue to the enlarged glands and up to the side of the head, had been so severe as to keep him awake, and necessitated his having hypnotics every night. He is now an out-patient, and for some weeks returned to his work as a painter. The ulcer on the side of the tongue healed, the induration became much less, and the glandular swelling softer. A very interesting, instructive, and important change took place in the hard mass of glands on the right side of the neck in this patient, commencing soon after the operation on the thyroid—viz., gradual and progressive softening. This went steadily on until there was great tension of the superficial surrounding tissues, and it was decided to incise the swelling and anticipate pointing and ultimate bursting of the enlargement. On freely opening it a large quantity of glairy mucoid fluid was discharged, which, on pathological examination by Dr. Wyatt Wingrave, was found to consist chiefly of mucin. The

inference, therefore, may be taken to be that the removal of the thyroid had induced a myxomatous degeneration in the mass of cancerous glands. Since this incision the mass of secondary growth in the glands has slowly diminished and much discharge has taken place ever since, containing pieces of broken-down tissue which have sloughed away. On inserting the finger a cavity can be felt where the mass of hard glands was.

DISCUSSION.

The PRESIDENT commented on the improvement to which the patients gave the most confident testimony. Such results were certainly entitled to very serious consideration.

Dr. DONELAN asked with reference to the right side what advantage was expected from the ligation performed there, in view of the fact that the circulation would be at once restored, as the greater sources of the blood-supply were left untouched.

Mr. STUART-LOW said the reason he undertook the treatment was because such cases were usually doomed and hopeless. He did not think radium would have had any effect upon such a large fungating tumour. The treatment had benefited the patient, as the pain had gone, he swallowed better, and his weight had increased by 1 st. 2 lb. He undertook this operation upon the theory that the thyroid had a driving effect on the tissues of the body, and probably a controlling action over other glands. It was now six months since the operation in one case, five months in another, and four months had now elapsed in the third case shown. The second man came with a huge mass of glands and a mass of cancer in the tongue. The glands gradually softened, and it looked as if the tumour would burst. When this was incised a large quantity of pure mucin poured out. So that probably, as a result of this operation, myxomatous degeneration had taken place, and now, the finger being inserted, a huge cavity was found where the mass of glands had been. The third case was given up, as being an inoperable carcinoma. Since this operation of hemithyroidectomy, however, he had gained in weight and undergone a progressive improvement.

Infiltration of Left Ventricular Band. ? Neoplasm. ? Tuberculosis.

By J. DUNDAS GRANT, M.D.

THE patient, a woman, aged 62, complained of hoarseness with occasional loss of voice while speaking, of one year's duration and apparently gradual development. There is extreme infiltration of the

tissues of the left ventricular band, which bulges irregularly so as to cover the whole of the left cord and the anterior part of the right one. It appears to dip occasionally between the cords so as to prevent their approximation. There are no enlarged glands, no expectoration, no signs of tuberculosis, but there is a tendency to it in her husband's family, he dying, however, of paralysis. A portion of the swelling was removed for microscopical examination, but the examination seems to indicate nothing beyond an inflammatory hyperplasia. It is proposed to remove a larger portion for further examination.

Dr. GRANT said that no tuberculous tendency could be made out, and that Dr. Wyatt Wingrave reported that it did not appear to be malignant. He would be happy to show the case again later when it became clearer.

Epithelioma of the Right Vocal Cord in a Man aged 60 ; Removal by Thyrotomy.

By J. DUNDAS GRANT, M.D.

THE patient was first seen by the exhibitor on September 24, 1909, complaining of increasing hoarseness of seven years' duration, which had got worse for six months. On examination of the larynx the right vocal cord was seen to be red and infiltrated, and just below it, in front of the right vocal process, there was a conical outgrowth measuring about 2 mm. at its base. A portion of the growth was then removed by means of the exhibitor's forceps, and a microscopical section was made by Dr. Wingrave, who reported that in parts it appeared very much like a squamous papilloma, but in others the variety, size, and shape of the cells were atypical. In other parts the characters of an early epithelioma were well shown. Solid cylinders of epithelium, with nest or pearl grouping of the cells and crowding of the original papillæ, were remarked. Heteromitoses were few and Altmann's granules absent. Lymphocytic infiltration was well marked, and there were nuclear fragmentation and wandering paranuclear spheres. With the assistance of Dr. Dan McKenzie, Dr. Grant performed thyrotomy, and found the right vocal cord infiltrated right up to its attachment to the arytenoid cartilage, a portion of which he removed along with the cord. The underlying cartilage was freely

scraped, considerable bleeding taking place from a spot above the middle of the side of the cricoid cartilage. The galvano-cautery was very freely used over the whole extent of the removal. Hahn's cannula was removed and replaced with an ordinary tracheotomy tube, which was removed next day. The patient was found to be able to swallow water next day, and was then fed by the mouth. He returned home in nine days. When last seen there was a grey, sloughy condition at the site of removal.

DISCUSSION.

Mr. DE SANTI asked if it was Dr. Grant's custom to cauterize these cases after excising the diseased area? His own opinion was that such a procedure was distinctly irritating and not necessary.

Dr. SCANES SPICER said that, as the malignant mass in this case had been referred to the Morbid Growths Committee, perhaps they would note whether or no the apparent site of origin, or of the point of maximum, or most advanced development, of the growth, had any relation to any prominent angle, ridge, or bump of any of the cartilages or bones of the throat over which the soft tissues played during functional activity—e.g., muscular or vocal processes of the arytenoids, &c. The frictions, stresses, and strains over such areas should be increased when these functional movements were greatly exaggerated—as, e.g., in abdominal breathing—and these might (if persistent and not too excessive) determine the commencement of cancer. He had been led to suspect such an intrinsic or endogenous source of mechanical irritation from the history, clinical appearances, and autopsies, of his own cases of cancer about the cricoid; and on examining the whole series of cricoid and œsophageal cancers in the Royal College of Surgeons Museum and elsewhere, there appeared to be no exception to the rule that the site of origin, or of maximum development, was such a special site of friction, stress, or strain. He had suggested as a working hypothesis that “belly” breathing not only produced increase of intrinsic mechanical irritation here—and also elsewhere in the body—but also a state of chronic auto-intoxication from a stagnant portal system, so that a primary cachexia ensued. He was continuing his own investigations on these points, but hoped all interested observers would join in.

Dr. GRANT replied that Dr. Wyatt Wingrave was very definite about there being an epitheliomatous lesion. The sections were available, and he would be glad to submit them to the Morbid Growths Committee. In answer to Mr. de Santi, he could only say it was a matter of opinion that, by cauterizing, one more effectually sealed up the lymphatics.

**Epithelioma of Left Vocal Cord in a Woman aged 58 ;
Removal by Thyrotomy.**

By J. DUNDAS GRANT, M.D., and DAN McKENZIE, M.D.

THE patient, a woman aged 58, was first seen by Dr. Dan McKenzie on account of hoarseness of eighteen months' duration, but which was stated to have come on in one night after an attack of vomiting. A small, irregular, reddish, sessile growth was found on the left vocal cord, somewhat rough but scarcely papillated on the surface, and with free movement of both cords. Dr. Dan McKenzie made a provisional diagnosis of epithelioma, in which Dr. Grant concurred; and this was confirmed by Dr. Wyatt Wingrave on examination of a portion of the growth removed, the tissues showing typical squamous epitheliomatous structure. With Dr. McKenzie's assistance, Dr. Grant performed thyrotomy, removed the whole of the vocal cord, and cauterized the site very thoroughly. The patient could drink next day and was discharged nine days after the operation, and when seen a week later showed merely a sloughy condition over the site of operation in the larynx. The after-treatment and the course were the same as in Dr. Grant's other case.

Partial Fixation of the Left Vocal Cord, presumably of twenty-one years' duration, in a Male aged 57.¹

By IRWIN MOORE, M.B.

TWENTY-ONE years ago a swelling, the size of a hen's egg, appeared at left side of neck—level of thyroid isthmus—accompanied by partial loss of voice. Patient attended the Golden Square Throat Hospital and saw the late Sir Morell Mackenzie and Dr. Wolfenden, who told him that one of his cords was paralyzed. Under the local treatment of iodine the swelling in neck gradually disappeared during the following five months; at the same time the voice also gradually returned. Had no further loss of voice or any other trouble till last May, when, after running in a hurry, he noticed a shortness of breath, followed by dryness of throat and some hoarseness of voice, which still continues. Also

¹ From the Throat Department, King's College Hospital.

patient complains of stiffness along left side of neck at level of cricoid cartilage. Has been married thirty-six years; thirteen children, three born dead at full term—viz., the fifth, seventh, and eighth. No history of syphilis. No aneurysm nor enlarged glands.

The case is shown for opinion as to whether we have to deal with a case of ankylosis of the crico-arytænoid joint or a lesion of the recurrent laryngeal nerve.

Dr. WATSON WILLIAMS said that as opinions were asked as to diagnosis, he thought it was ankylosis, for the vocal cord was fixed even further out than the position of ordinary abduction, a position it would not occupy if it were a lesion of the recurrent laryngeal.

Paralysis of the Right Vocal Cord in a Case of Myotonia Atrophica.

By H. CLAYTON FOX, F.R.C.S.I.

THIS case was shown at a previous meeting of the Section, when it was deemed advisable to have the opinion of a neurologist.

Dr. Frederick Batten had very kindly provided the following notes: For many years the patient has experienced difficulty in relaxing his grasp, a trouble in first starting to walk in the morning, and in going downstairs. In December, 1907, he had an acute illness, diagnosed as congestion of the liver, which was immediately followed by a difficulty in speaking. Family history: Father died aged 64; no signs of muscular wasting or myotonia. Mother had always had a difficulty in relaxing her grasp, but this lessened as she grew older; she died, aged 82, of senile decay. Other members of the family healthy. Present condition: Patient has the myopathic facies; pupils are equal and react well to light and accommodation; ocular movements good; no ptosis or nystagmus; the face is smooth and expressionless. There is great weakness of the orbicularis palpebrarum and some loss of power in the orbicularis oris; the temporal muscles are active, but diminished in bulk; the masseters, tongue and palate are normal; both sternomastoids are completely atrophied, and there is some wasting of the upper part of the trapezii; movements of the head and neck are performed with fair power; both forearms are flabby and slightly wasted, the flexors and extensors participating to an equal degree; all

movements of the wrists are fairly good; no atrophy of the small hand-muscles. When the patient is asked to grasp an object he does so perfectly, but experiences difficulty in relaxing his hold. Trunk muscles normal; slight lordosis in the lumbar and dorsal regions; the vastus internus and externus of both limbs are completely atrophied; the calf muscles and the anterior tibial group are hypertrophied; the knee-jerk is absent on the left side and difficult to elicit on the right; ankle-jerks absent on both sides; plantar responses flexor; no sensory changes; in walking the feet are unduly elevated. Patient's method of rising from the recumbent position resembles that met with in pseudo-hypertrophic paralysis. The right vocal cord is fixed midway between the cadaveric and phonatory positions; there is no evidence of any lesion of the vaso-accessory, either centrally or peripherally.

The case is shown to elicit opinions as to whether the laryngeal lesion be a part of the general condition or otherwise.

DISCUSSION.

Dr. FREDERICK E. BATTEN said that from the neurological point of view the case was of interest because it belonged to a somewhat rare group, which had been described under the term "myotonia atrophica." The striking features of these cases were the weakness of the facial muscles, absence of the sterno-mastoids, and wasting in other muscular groups of the limbs. In association with that, such patients had a peculiar action of their muscles, very much like that seen in Thomsen's disease, so that they were unable to relax their muscles. A point of interest to laryngologists was that two of the other cases which had been described by Continental observers had had paralysis of the vocal cords. One was described by Nonne and the other by Siemerling. It was at present doubtful to which group these cases should belong, as the pathology had only been worked out in a single case by Steinert. In that case the muscles showed a condition which one associated with a myopathy, but there was also some degeneration of the posterior columns of the spinal cord. In several of the cases which had been described the knee-jerks were absent. They could not, however, be regarded as cases of tabes.

The PRESIDENT said it did not seem to be quite a typical case of recurrent laryngeal paralysis; the cord was nearer the middle line than would be expected in pure recurrent laryngeal paralysis after such a long period. He thanked Dr. Batten in the name of the Section, and hoped other distinguished Fellows in other departments would follow his example.

Mr. CLAYTON FOX, in reply, agreed that the case looked like myopathy or ankylosis, rather than a neuropathy, from the fact that the cord was not shortened and the arytaenoid on that side was not sunken downwards, inwards and forwards. He thanked Dr. Batten for his remarks.

**Swelling on Left Side of Neck, accompanied by Dyspnœa, in
a Male aged 28.**

By NORMAN PATTERSON, F.R.C.S.

THE swelling began in August, 1909. In September it burst, pus escaped, and the dyspnœa was relieved. There is now a sinus along which a probe can be passed in an upward direction for some considerable distance. The larynx presents a globular swelling in the region of the left arytenoid cartilage, which disappears to a large extent during phonation. There are signs of tuberculosis of right apex. Opinions are asked as to the diagnosis of the laryngeal and cervical condition.

The PRESIDENT said it looked as if there was a limited patch of œdema over the cartilage of Santorini, which was drawn in during inspiration.

Case of Acute Pemphigus of the Larynx in a Woman aged 40.

By H. J. DAVIS, M.B.

THIS case is a very interesting one. The patient has been attending the skin department under the care of my colleague, Dr. Abraham, who recently asked me to see her owing to onset of dysphagia and loss of voice of seven days' duration. She is suffering from a severe form of pemphigus vegetans, and is now an in-patient in the hospital. In addition to bullæ all over the body, some of which are healing and others recent, the nasal mucous membrane, the lips, tongue and pharynx are also affected; in the fauces the affection somewhat resembles secondary syphilis. But it is the interior of the larynx which presents so peculiar an appearance; the surface is raw and blistered; there is no œdema, but the cords, which meet and move perfectly, are a brilliant crimson. The surfaces are being dusted with orthoform.

DISCUSSION.

Mr. CRESSWELL BABER said the case reminded him of a case of pemphigus in the mouth and throat which he showed before the Laryngological Society six years ago.¹ The patient was aged 69. There was no affection of the skin

¹ *Proc. Laryngol. Soc. Lond.*, 1903-4, xi, pp. 64 and 181.

when the patient was shown, but afterwards the disease appeared on the integument, and he died of exhaustion some months later. He had large doses of arsenic internally, but without beneficial effect.

Dr. D. R. PATERSON had under observation at the present time a patient who was in the immediate charge of an ophthalmic colleague. She showed very well the essential atrophy of the conjunctivæ with shrinkage of the globe, due to connective-tissue change following on bullæ formation, met with occasionally in such cases. Bullæ with milky contents were once or twice seen on the soft palate, but the appearances were mostly whitish plaques on the pharynx, epiglottis, and over the arytaenoid region, and those were rarely absent. In the interior of the nose were areas of superficial ulceration with crusts, and there was evidence of some pus around one middle turbinal. The disease had affected the scalp and the limbs, had persisted for years, and was supposed to have begun in the eye. The patient was definite, however, in her statement that it was in the throat that she first felt trouble. It seems not unlikely that all such cases begin, like Mr. Baber's, in the mucous membrane.

Dr. DAVIS, in reply, said he did not think the condition began in the mouth and spread to the skin.¹ The patient had been attending the West London Hospital for a long time, and she had now enormous patches of pigmentation all over the body. Her larynx had only recently become affected. When he first saw her he thought there was a possibility that the condition of the mucous membrane might be due to the arsenic which she was taking. But that was not so. Pemphigus vegetans was a disease invariably fatal.

Demonstration of Hay's Pharyngoscope.

By H. J. DAVIS, M.B.

THIS instrument, a very ingenious one, was first brought to my notice by Dr. Holbrook Curtis, of New York, in July last. Illumination of the tiny lamps is obtained either by means of a transformer or, better still, a small dry battery. By rotating the periscope the entire post-nasal space, the Eustachian tubes, and the interior of the larynx are brought successively into view. In a suitable case, as in the patient exhibited, an excellent view of all these parts can be obtained.

¹ P.S.—November 16: The patient has improved considerably. The exhibitor finds that he was in error when he stated that the throat affection was secondary to the skin lesions. The patient now states that her fauces were affected ten months before the cutaneous bullæ appeared, but the affection of the larynx is of recent date.

Soft Foreign Body in the Bronchus.

By D. R. PATERSON, M.D.

A CHILD, aged 6, was brought on September 1, 1909, with the history of having, six weeks previously, whilst eating "monkey" or ground nuts (*Arachis hypogæa*), got one "down the wrong way" and had a severe choking-fit, which only ended by its being displaced. From that time wheezing and attacks of cough till she nearly choked were present, and she had been ill, with raised temperature, pain in right side of chest, and coughing up some matter. The child looked ill and had lost flesh, had a temperature of 101° F. and a pulse of 120. Frequent dry, irritable cough, almost "croupy" at times, and stridulous breathing were noted. Sibilant and sonorous rhonchi were audible over the chest, though on the right side the chest movements and breath sounds were diminished. At the same base, fine râles were heard, as well as a whistling sound during whole of expiration. Under chloroform a telescopic tube (7 mm.) was passed through the glottis down the trachea, and the nut was seen a short distance from the bifurcation in the direction of the right main bronchus, just below the origin of the branch to the upper lobe, the opening of which, narrowed from swollen mucous membrane, was inspected. The body was firmly fixed and filled the lumen except at one point, through which the respiratory air whistled and muco-pus escaped. The surrounding mucous membrane was swollen and red. The nut was grasped by a telescopic forceps having the fenestrated "bean" point, and, care being taken to avoid crushing it, was removed without breaking. It consisted of half the kernel. Examination showed the bronchus clear and a patch of ulceration where the body impinged on the wall. On auscultation the air entered freely and the whistling sound had gone. The child made an uninterrupted recovery.

The case belongs to the important category of soft foreign bodies which are always most urgent and demand prompt interference. They are dangerous from the complications which rapidly ensue and from the liability of the foreign body to break during extraction.

Two Cases of Malignant Disease of the Neck, undergoing Treatment by Radium.

By WILLIAM HILL, M.D.

Case I.—Male, aged 38, came to St. Mary's Hospital in July last, complaining of marked dysphagia; he was only able to swallow liquids. There was a very large swelling on the left side of the neck, occupying the anterior and posterior triangles, and extending above to the mastoid region, zygoma and lower jaw, and below to the clavicle; its anterior limit was near the middle line of the neck, and it extended posteriorly to within two inches of the spinous processes. It was extremely hard, and an outlying portion, removed under local anæsthesia for microscopical examination, proved it to be an endothelioma. The patient had slight facial paralysis and complete recurrent nerve paralysis, the left cord being fixed. By œsophagoscopy it was seen that the lumen of the upper two inches of the gullet was markedly narrowed and crescentic in form, the wall of the tube projecting into the lumen in the left side and presenting two well-marked ridges, one above the other; there was no ulceration, and the endoscopic appearances were suggestive of invasion from the tumour in the neck. During the last four months the man had had five separate applications of radium, amounting to something over one hundred hours in all. Dr. Finzi kindly treated the case with his tube containing 50 mg. of pure radium bromide, screened with a lead casing 1 mm. thick. One application of seventeen hours' duration had been made within the gullet, the tube being inserted under direct vision by means of the œsophagoscope (Brünings' largest tube-spatula). The other applications were made to the cutaneous aspect of the growth. The applications were made at intervals of from three to five weeks, in order to allow reaction to subside. The dysphagia was relieved early in the treatment, and had now entirely subsided, but the œsophagus was not quite normal on endoscopic examination; the facial paralysis had nearly disappeared, but the vocal cord was still fixed; the most striking change was in the size of the growth, which has been reduced to at least one-eighth its former dimensions; there is no obvious bulging of the neck, but the remains of the growth can be felt as a small, flat, hard thickening below the margin of the jaw near the angle. The case will be shown again from time to time.

Case II.—A middle-aged man with a similar large tumour, also on the left side of the neck. There was no dysphagia and no obvious lesion

seen by œsophagoscopy, but there is paresis of abduction of the left cord. The man had only recently been referred to St. Mary's Hospital by Dr. Batten, of the Central London Sick Asylum, and had only one application (50 mg. for twenty-two hours) two days previously. The surface of the tumour was extremely red as the result of this application. A cast of the neck had been taken, and this, together with the patient, would be brought before the Section again from time to time, whether the result were favourable or otherwise.

Epithelioma of Nasal Vestibules and adjacent Areas ; recurrence after Surgical Removal ; Radium Treatment commenced.

By WILLIAM HILL, M.D.

C. W., AGED 48. This man was shown at the June meeting of the Section with an ulcerated malignant growth on the floor of the left nasal vestibule, and with tumefaction of the adjacent parts of the columella, left ala, and lip. When previously shown it was provisionally described as probably rodent ulcer. A subsequent microscopical examination, however, showed it to be a true epithelioma. The malignant area was excised by an incision half an inch wide of the apparent margin of the growth, extending right down to bone. Recently there had been signs that the removal had been imperfect, and Dr. Finzi had, a week previously, applied his radium tube for three hours between the lip and the alveolus, and for fifteen hours on the facial aspect of the diseased area. The case will be shown again from time to time.

An Improved Direct-vision Laryngoscope for Endolaryngeal Operations.

By WILLIAM HILL, M.D.

THIS new instrument was employed to demonstrate the laryngeal condition of a middle-aged man who had recently been operated on by the exhibitor for epithelioma. The right arytenoid cartilage, right ventricular band, right cord and adjacent soft subglottic tissues had been removed by thyro-fissure. There was now laryngeal stenosis suggestive of recurrence. (Since the meeting it has been found necessary to remove the entire larynx.)

Laryngological Section.

December 3, 1909.

Dr. J. DUNDAS GRANT, President of the Section, in the Chair.

Peculiar Erythema of the Left Palate in a Man aged 41 who had been a considerable smoker.

By J. DUNDAS GRANT, M.D.

THREE months ago the patient discovered a redness of his palate when suffering from a slight sore throat. There can be little doubt, however, that the condition is of much longer standing in view of the white sclerotic state of the mucous membrane covering the hard palate. The tongue and larynx are normal. The patient has smoked about 2 oz. of tobacco a week in a short pipe.

The exhibitor brings the case forward for diagnosis. He is inclined to think that it is an early buccal manifestation of a dermatosis, but there is as yet no sign of disease of the skin.

DISCUSSION.

Mr. BARWELL thought it was either petechial or of the nature of a capillary nævus, as it did not fade on pressure.

Dr. H. J. DAVIS regarded it as a septic condition due to the bad teeth—pyorrhœa alveolaris.

Dr. DAN MCKENZIE said there was very marked pyorrhœa, and the patch on the palate seemed to him to be excoriation rather than congestion and dilatation of the capillaries. Consequently he agreed with Dr. Davis.

The PRESIDENT (Dr. Dundas Grant) said he had used the term "erythema" rather loosely. The only condition he had seen like it was a case of lichen planus, which was shown at a meeting of the German Laryngological Society at Heidelberg. He did not think the condition of the teeth sufficient to explain the appearance, but in any case they should be looked to.

Secondary Specific Pharyngitis in a Young Woman.

By J. DUNDAS GRANT, M.D.

THE patient, aged 22, was sent to Brompton Hospital as presumably a subject of tuberculosis. When referred to the exhibitor he was able to agree with the colleague to whom she was sent, in his opinion that the condition of the throat was really one of secondary specific nature. On both pillars of the fauces there was superficial ulceration of symmetrical nature, rather higher in colour than a tuberculous lesion would be. It was of about two months' duration, and was more marked on the left side, where apparently it had commenced. In the left sub-maxillary region there was an enlarged gland, which seemed to indicate that the left tonsil might have been the seat of an accidental primary inoculation. No history was obtainable, and there was complete absence of rash and of post-cervical glandular enlargement. The patient is an exceptionally healthy-looking person. The examination of a scraping from the diseased surface, by Dr. Bryant, revealed the *Spirochata pallida*.

Dr. DONELAN said this case illustrated the very great value of the bacteriological test in these doubtful cases. He had a very similar case seven or eight years ago, in which, owing to the surroundings of the patient, a young lady, there was great difficulty in expressing an opinion or in ordering antisiphilitic treatment, even to clear up the diagnosis. As it was, the mere allusion to syphilis as one of the possible solutions led to the removal of the patient to the care of a colleague, who ultimately cured her by mercury and iodides.

Frontal Sinus Disease, with Necrosis, in a Man aged 33.

By W. H. KELSON, M.D.

THE patient, a tailor, contracted syphilis in 1905. In 1907-8 he attended an ophthalmic hospital for defective vision. In December, 1908, he came to the London Throat Hospital with pus in the left nostril, and a radical operation was performed on the left maxillary antrum by Mr. Seccombe Hett. Four days after that great swelling occurred in the frontal region on both sides. Three days later Mr. Hett operated again,

when extensive necrosis of the anterior walls of the frontal sinus was found and large sequestra removed. The case then came under my care; since then small portions of dead bone have been taken away on several occasions, and a node has appeared on the left side of the mandible.

Addendum.—The patient has since had a fit and did not recover consciousness for about half an hour. No paralysis is present. An area of bare bone has also become apparent on the *posterior* wall of the sinus on the left side.

A Case of Ulcer of Doubtful Nature on the Left Arytænoid Cartilage in a man aged 63.

By H. BETHAM ROBINSON, M.S.

THERE is a flattish ulcer creeping from the inner side of the arytænoid cartilage on to the posterior part of the cord; on the upper surface of the cord are some heaped-up granulations over its posterior two-thirds, but not involving the free edge, which is sharp and well defined. The cord is generally congested. There is no evidence of infiltration, and mobility is normal. The right cord is not altered. There are no enlarged glands, no pain, no physical signs in the chest, and no bacilli in sputum. In February last he had hoarseness and slight pain. He was sent to me for an opinion in June, when he was still hoarse, but had no other inconvenience; practically there has been little change in the laryngoscopic view since. In July he was operated on for glaucoma. The suggested diagnosis is that it is a senile tuberculous ulceration; the length of duration and treatment seem to have excluded syphilis and carcinoma, apart from the local appearances.

DISCUSSION.

Dr. WATSON WILLIAMS suggested that complete and absolute rest for a fortnight should be tried. His own view was that it was most likely to be tuberculosis, but it was lacking in definite signs, and he did not wonder that Mr. Robinson found difficulty in arriving at a diagnosis.

Mr. DE SANTI also regarded the case as senile tuberculosis. At first he thought it might be malignant, but the length of time it had existed, the

free mobility of the part affected, and the absence of any affection of the glands put malignant disease out of the question.

The PRESIDENT said his opinion coincided with Mr. de Santi's; the peculiar greyness of the floor of the ulcer and the languid purplish colour of the edges were what one often saw in tubercle. It was exceptional in that it was so limited to one part. Possibly the rest of the larynx would later be involved. He reminded Mr. Robinson that several members had often found galvano-caustic treatment successful in similar cases.

Mr. ROBINSON replied that the patient had already had a considerable amount of rest, and there was definite improvement. He agreed that it was senile tubercle.

Chronic Laryngitis in a Man aged 26. ? Prolapse of Ventricle on Right Side.

By H. BETHAM ROBINSON, M.S.

THE patient came on September 8 with some loss of voice and hoarseness of two months' duration; he had then chronic pharyngitis with catarrh of both vocal cords. On October 10 the swelling over the right cord was first noticed. At the present date there is to be seen over the anterior two-thirds of the right cord a red fold, which in shape might be likened to the plica semi-lunaris; this is free from the vocal cord, but is connected with the edge of the ventricular band. The posterior part of the right ventricular band seems a little more convex than on the left side. At times the mobility of the right cord appears a little impaired. Both cords are catarrhal, but not ulcerated. No physical signs in the chest.

DISCUSSION.

The PRESIDENT regarded the case as tubercle, but admitted it was open to question.

Dr. H. J. DAVIS said he did not know why Mr. Robinson called it "prolapse of the ventricle." He would have called it "a swollen ventricular band." Prolapse he regarded as a more extensive condition.

Mr. HERBERT TILLEY agreed with Dr. Davis; he thought it was probably a tuberculous lesion. The direct method with a good light would have given more information about the exact place from which that swelling started. The

light in the examination room was not good, and when he came to examine the patient the latter had become somewhat irritable. It seemed to be rather an œdematous condition on the anterior part of the right vocal cord. Possibly the galvano-cautery would give relief.

Mr. HORSFORD suggested that the swelling, which was seen posteriorly behind the œdematous portion, was subglottic, not affecting the vocal cord itself, and thus further supporting the theory of its inflammatory nature.

Dr. JOBSON HORNE thought the term "prolapse of the ventricle" was being used too freely. It was a very rare condition; and many of the so-called cases of prolapse were growths protruding from the ventricle. He had not examined the patient exhibited, as he had already been examined so many times that evening and was a little tired.

The PRESIDENT said he thought the term "prolapse" in this connexion was the remains of a tradition which had been handed down. Gouguenheim said the cases of so-called prolapse were generally out-growths, usually tuberculous, from the ventricle.

Mr. ROBINSON, in reply, pointed to the query-mark in the title. It was evident therefrom that what he meant was a pouting out of the mouth of the ventricle.

Hoarseness of One Month's Duration in a Woman aged 60.

By GEORGE C. CATHCART, M.D.

THE patient came last week complaining of hoarseness and loss of voice of a month's duration. On laryngoscopic examination the whole larynx was seen to be inflamed and red. There was very great difficulty in getting a view on account of the patient's intense nervousness, but it was possible to make out that there was almost complete occlusion of the anterior two-thirds of the larynx and a narrow chink between the vocal processes in which were numerous crusts. The case is shown to elicit the opinion of members as to what the condition really is, and the best method of treatment.

DISCUSSION.

The PRESIDENT said there was a symmetrical lesion of the larynx involving chiefly the ventricular bands. The picture seemed to be that of tuberculosis, but there was nothing but the picture to support that diagnosis.

Dr. WATSON WILLIAMS said he wondered whether it could be called glandular laryngitis. It was peculiarly symmetrical, and it did not look to him like tuberculosis; it seemed a more simple condition.

Dr. CATHCART, in reply, said he could not find any evidence of tuberculosis in the patient, nor was there any family history of the disease; she had no cough, nor had she been losing flesh. There had been no other symptoms, except hoarseness and loss of voice.

Tuberculosis of Epiglottis treated by Amputation of Diseased Portion.

By HERBERT TILLEY, F.R.C.S.

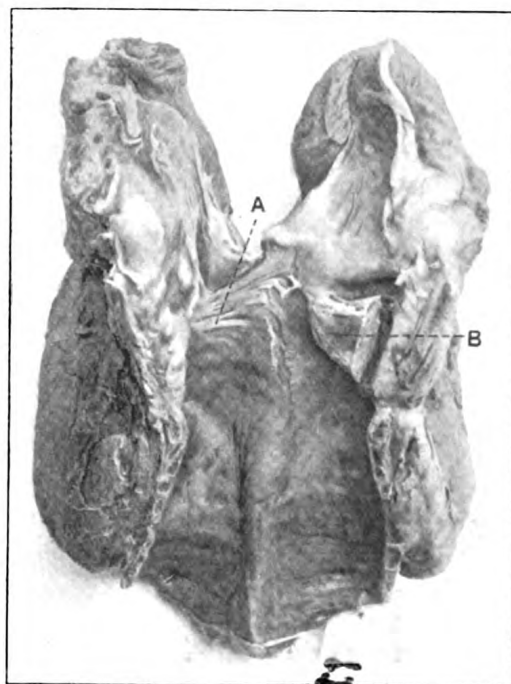
MR. L., aged 43. Seen March, 1907, on account of "slight pain on swallowing," "losing flesh," and frequent profuse "sweating at night." Physical signs of pulmonary tuberculosis were found, and evening temperature for a fortnight varied from 99·6° F. to 100·8° F. The tip of the epiglottis was swollen and ulcerated, and there was a slight extension of this condition downwards into the right aryepiglottic fold. The diseased region was cocaineized and removed with a large-sized pattern of Lake's epiglottis punch forceps, and the patient then spent four months in the Ventnor Royal National Hospital. He rapidly recovered, and the stump of the epiglottis still presents a healthy scar. The patient weighs 13 st. 10 lb. as against 11 st. 3 lb. before operation.

Mr. HAROLD BARWELL said, in connexion with that successful case, that, although tuberculosis attacked the epiglottis very late, when the rest of the larynx was extensively involved, there was a distinct group of cases in which the epiglottis was attacked first. This generally occurred where there was chronic disease of the lung, and the affection of the larynx was also chronic. It started at the tip of the epiglottis, and the ulceration extended downwards. If the disease could be removed *en masse* with punch forceps while there was healthy tissue between the ulcer and the glosso-epiglottic folds, there was a good chance of cure. Lake's forceps were of great advantage here, as they took off the affected part of the epiglottis at one bite. He (Mr. Barwell) believed he had improved those forceps by making the lower or cutting edge flatter, so that it did not leave a projecting horn on each side of the epiglottis, such as was produced by the circular punch.

When is Cancer Cured? Larynx of a Man, aged 78, whose Right Vocal Cord was removed for Epithelioma in September, 1896.

By HERBERT TILLEY, F.R.C.S.

SINCE the above date the patient enjoyed good health until a month ago, when slight difficulty of breathing occasionally occurred; this became distressing about November 15. I saw the patient in consultation on



Mr. Tilley's case. **A**, scar of operation performed thirteen years ago; **B**, epitheliomatous growth on left vocal cord—the cross-section indicates portion of growth removed for microscopical examination.

November 18; his doctor performed tracheotomy on November 19, and nine hours later the patient died. The cicatrix corresponding to the original right vocal cord and arytenoid is still quite healthy, but a

well-developed epitheliomatous ulcer occupies the greater part of the *left* vocal cord.

The case was seen by Sir Felix Semon in consultation with me before the operation in 1896; he confirmed the diagnosis of epithelioma, which was proved by the microscope after operation. The patient was shown at the Laryngological Society some five years after operation.

DISCUSSION.

Mr. TILLEY said the patient was shown at the Society some five years after the operation, and he had not seen him again until a few days ago. When recently called to the patient the breathing was accompanied by much stridor, and it was obvious that laryngeal obstruction was present.

The PRESIDENT said there could be no doubt about the diagnosis.

Dr. H. J. DAVIS said it seemed that the patient had had epithelioma once, and was cured of it ten years ago. Then he had it a second time ten years afterwards, and he did not think the one was the cause of the other. This point was one of interest and importance.

Mr. DE SANTI agreed that this was not a true recurrence, but really a fresh outbreak. The bringing forward of such cases was both important and interesting. He had had a patient from whom he removed the mammary gland for carcinoma many years ago. She did very well in the ordinary way, and was watched for years afterwards. Eleven years later she came back to hospital with carcinoma of the liver. She died, and a post-mortem was made, and no malignant disease was found elsewhere than in the liver. He did not call that a recurrence. There must have been some peculiar predisposition to malignant disease in the patient, and one was entitled to say that the operation for the breast and axillary glands was a cure.

Dr. STCLAIR THOMSON said there were a few cases on record in which a malignant growth on one vocal cord was supposed to have infected the opposite cord: such cases had been reported by Mr. Newman, Mr. Butlin, and Sir Felix Semon. The present case showed it might have been a fresh outbreak, and those who were investigating the pathology of cancer might like to hear of the case. He had a case in which a vocal cord was removed and epithelioma occurred on the tongue on the opposite side.

Dr. MIDDLEMASS HUNT said the case reminded him of one he had had which ended fatally. He was at first proud of it because for five years he regarded it as a perfect result. One cord had been removed, but in five years there was a gradual thickening of the other cord. The patient was operated upon for that, but not successfully because of the rapid extension to the external parts.

Dr. WATSON WILLIAMS suggested that the specimen should be referred to the Morbid Growths Committee, not because there was any reasonable doubt that the case was one of epithelioma, but on account of the special interest and importance of the specimen, which would be enhanced by a report from the Committee.

Dr. JOBSON HORNE inquired whether the original microscope specimen obtained in 1896 had been exhibited, and, if not, whether the specimen, together with the present macroscopic specimen, might be referred to the Morbid Growths Committee to report upon, and so complete the record of the case.

Complete Stenosis of Larynx left after Diphtheria and Tracheotomy, in a Boy when aged 1 year and 2 months ; completely cured by Repeated Intubations spread over Three Years.

By STCLAIR THOMSON, M.D.

THIS little boy, Herbert B., was admitted in September, 1906. Three weeks before admission tracheotomy had been performed for diphtheria. On admission it was found that he was absolutely voiceless; there was no respiration through the larynx, but he breathed entirely through a well-done low tracheotomy. By direct laryngoscopy it was seen that the cords were intact, but with a web immediately below them completely closing the glottis. Under chloroform this diaphragm was broken down with a probe and the smallest-sized intubation tube was inserted. The tracheotomy tube was removed, but the wound in the neck was kept open by wearing a rubber obturator such as is used in the alveolar drainage of maxillary sinusitis. The wearing of the intubation tube or the tracheotomy tube was alternated from October, 1906, up to the summer of 1909. The child learnt to speak in the intervals of being intubated, but the intubation tube had soon to be replaced owing to gradually returning stenosis immediately below the cords. The tracheotomy tube was blocked up about three weeks ago, and finally removed a fortnight ago.

It will be found that the boy, who is now aged about 4 years 4 months, speaks freely with a strong, clear voice. The tracheotomy wound is closed. His breathing is a little stridulous on exertion, and it is found that this is due to the fact that the left vocal cord is

completely fixed in the cadaveric position, or slightly internal to it; this is evidently due to cicatrization around the arytaenoid joint. If handled gently, the small boy shows his larynx quite well by the indirect method.

This case shows that laryngeal stenosis from diphtheria may occur even although a good, low tracheotomy has been carried out. It is also claimed that the case shows the success of treatment in even complete occlusion of the glottis by means of tracheotomy and intubation. The only objection is the duration of treatment, but with patience and perseverance a result is obtained which is seldom equalled and never exceeded by such methods as laryngo-fissure or laryngo-tracheostomy.

DISCUSSION.

Mr. HERBERT TILLEY said that at present there was in University College Hospital a little boy who had been relieved in the same way by repeated intubation of post-diphtheritic laryngeal stenosis. In this case the tracheotomy had been performed high up. He agreed with Dr. Thomson that intubation was a better method than laryngo-tracheostomy.

Dr. STCLAIR THOMSON, in reply, reminded members of the debate at Belfast, in which much was learned from their American colleagues.

A Case of Probable Malignant Disease of the Larynx in a Woman aged 47.

By HAROLD BARWELL, F.R.C.S.

THE patient is the mother of six children, of whom five are alive and healthy, and one dead as the result of an accident; no miscarriages. Pain in the throat, especially on swallowing, began eleven months ago, and at the same time a lump was noticed under the jaw. The dysphagia has got worse and now radiates to the right ear. There are some hard, fixed glands behind the angle of the jaw on the right side. A large mass fills the right half of the larynx and conceals the right cord; the left cord is normal and the speech is unaffected, showing that the right cord is not involved. The mass is covered by conspicuous white elevations. The growth has not spread on to the pharynx. The symptoms have somewhat improved after a fortnight's treatment with potassium iodide and mercury.

The exhibitor would welcome opinions as to operation, especially as to whether anything short of a complete laryngectomy might be advisable in view of the healthy condition of the cords.

DISCUSSION.

Mr. ATWOOD THORNE asked how long the case had been under treatment, and what was the condition of the case a few months ago. He thought that it would have been easily operable earlier.

Mr. DE SANTI said the fact of the vocal cords not being affected was in favour of doing some form of laryngectomy. Thyrotomy was essentially the operation for the intrinsic variety. The only point in the case was as to whether a hemi-laryngectomy or a complete laryngectomy should be done. He recommended splitting the thyroid cartilage, examining the extent of the disease, being prepared accordingly to perform either hemi- or complete laryngectomy and to remove the glands. Personally, he felt fairly confident that complete removal of the larynx would be necessary, and the patient should have the situation carefully explained before operation.

Dr. H. J. DAVIS said it looked like a fungating gumma. It did not follow because there were enlarged glands in the neck that it was carcinoma.

Mr. BARWELL replied that he did not see the case until two or three weeks ago. He had hoped to obtain the opinion of the Section as to the advisability of operation, and whether any other course than complete laryngectomy would hold out any prospect of cure.

A Series of Skulls Demonstrating Variations in the Relations of the Sphenoidal Sinus and of the Spheno-ethmoidal Cell.

By W. S. SYME, M.D.

THESE skulls are culled from a total of 250 examined, and are examples of the more or less abnormal relationships which may exist: (1) between the two sphenoidal sinuses; (2) between the spheno-ethmoidal cell and the sphenoidal sinus; (3) between the sinus and the optic foramen on its own and on the opposite side; (4) between the sinus and the carotid tract on its own and on the opposite side; (5) between the spheno-ethmoidal cell and the optic foramen. Some of the specimens also show the formation of well-marked septa in the sphenoidal sinus.

**Specimen of Goitre and Portions of Larynx and Trachea
from a Woman aged 37.**

By JAMES DONELAN, M.B.

THE goitre caused first left recurrent paralysis, then right, with inspiratory dyspnœa. High tracheotomy was performed by the exhibitor, who was called in on the emergency. The patient died from tachycardia after the dyspnœa had been relieved.

Dr. FITZGERALD POWELL thought it probable that the patient died as the result of pressure on the recurrent laryngeal nerves. The patient must have got adductor spasm, and it opened up the question as to whether, where one found disease of the thyroid gland causing pressure on the recurrent nerves, one should not operate at once because of the great danger. Recently he had the case of a lady with a goitre on one side pressing on her recurrent nerve. She got adductor spasm, dropped down, went black in the face, and became unconscious. This occurred on several occasions. She was cured by operation: the right lobe being removed. Several cases had come under his notice in which death had occurred rapidly as the result of pressure by goitre. In all probability if the thyroid, or part, had been removed in this case the patient would be alive now.

Case of Laryngeal Growth.

By P. R. W. DE SANTI, F.R.C.S.

THIS patient, a woman between 40 and 50, was shown at the meeting of the Laryngological Section, May, 1909,¹ with a tongue-like growth attached to the anterior commissure and right vocal cord. This growth was movable, soft, and in colour like a soft fibroma. A piece had been removed and reported to be microscopically an angioma. An important factor was that the whole of the right vocal cord was uniformly red and somewhat thickened. The growth was considered by members to be a soft fibroma. I removed it with endolaryngeal forceps, but since removal there has been a recurrence, and microscopic

¹ *Proceedings*, 1909, ii, p. 140.

sections of the growth reveal masses of giant cells and one tubercle bacillus. The woman has physical signs now of pulmonary tuberculosis, and there is considerable interarytænoid swelling and swelling also of the right vocal cord. The question is, was the growth shown in May a "tuberculoma" or a simple soft fibroma, and the now tuberculous condition an infection that has taken place only since the patient was last shown? I invite opinions as to diagnosis and treatment.

DISCUSSION.

Mr. BARWELL said it was a peculiar and instructive case. He did not think anyone suggested last time that it was tuberculosis, but now the appearance was decidedly suggestive of that disease.

Dr. R. H. HODGSON said he thought that in this case the tuberculosis of the pharynx was extending into the lungs, and he suggested that Mr. de Santi should give pure ether, with hexamethylene tetramin in it, by inhalation. Although the tuberculosis might have advanced far into the lungs, this treatment would give unexpectedly good results. It must be clearly understood that whatever be the drugs given, there must be absolutely no re-breathing of the same.

Dr. H. J. DAVIS said he thought at first that it was a cyst, as it was so translucent.

Mr. DE SANTI, in reply, said there was never a cyst: it was always a solid œdematous mass.

Cases of Malignant Disease Treated by Radium.

By WILLIAM HILL, M.D.

Case I.—The patient, a man, with a malignant tumour of the neck, was shown at the last meeting;¹ and is now again brought forward to show the decrease of the tumour after the continuous application of 50 mg. of pure radium bromide for twenty-two hours.

Case II.—A private patient, male, with carcinoma of gullet. Six intra-œsophageal applications of radium have been made since July; before treatment the patient could only swallow liquids and purées; but now he can "eat almost anything," to quote the patient's own words.

¹ *Proceedings*, p. 25.

Removal of the Frontal Bone for Septic Osteomyelitis.

By CHARTERS J. SYMONDS, M.S.

THE infection followed chronic suppuration of both frontal sinuses, and existed before operation. In a series of operations the frontal bone was removed, together with a part of the sphenoid, the roof of the right orbit and its inner wall, and the orbital ridge. On the left side part of the orbital ridge was left.

The case was shown as an example of a rare complication, and as a recovery from a grave condition of infection. The other point of interest was the reconstruction of the greater part of the frontal bone and the consequent restoration of the elevation of the forehead. On the left side the frontal sinus had been operated upon by the method of Watson Williams with no perceptible scar.

Two Cases of Hoarseness.

By E. W. ROUGHTON, F.R.C.S.

Case I.—A man, aged 26, who has been hoarse since infancy. This was attributed by the patient to a scald on the neck. He has complained of pain in the throat and increased hoarseness for the last three months. There is a raised ulcer occupying most of the right vocal cord, the mobility of which is impaired. It is thought to be malignant.

Case II.—A woman, aged 42, who has complained of cough and hoarseness for five or six years. The vocal cords are reddish and swollen, the ventricular bands are swollen, and there is much thickening in the interarytænoid region. There is no ulceration. No sign of phthisis in chest and no tubercle bacilli in the sputum. The case is exhibited with a view to eliciting opinion as to diagnosis and treatment.

DISCUSSION.

Dr. H. J. DAVIS said he regarded the first case as malignant, but the patient was young for that.

Mr. TILLEY said he would have thought the diagnosis rested between tubercle and syphilis, the latter being more likely.

Dr. FITZGERALD POWELL said in his opinion the second case was one of chronic laryngitis with interarytænoid pachydermia. He could see no evidences of tuberculosis, and believed it to be a simple chronic inflammatory affection. There was, he thought, some nasal obstruction, and this should be put right, when probably the patient would be cured with the ordinary treatment.

The PRESIDENT regarded it as pachydermia of the larynx in an alcoholic subject.

Model by which the Variation in Effect of "Back" and "Belly" Breathing on the Stresses, Strains, and Frictions in the Throat and Larynx, more especially of the Cricoid Cartilage on the Spinal Column, and also the Transverse Axis of Respiratory Rotation of the Cricoid on the Thyroid Cartilage, can be illustrated.

By R. H. SCANES SPICER, M.D.

THIS (home-made) model is shown to bring to the early knowledge of those specially interested in the causation and treatment of derangements of the upper air and food passages certain conceptions and conclusions which the author has arrived at in continuing his investigations on the action of intrinsically arising (autogenetic) mechanical forces in the human body in the genesis of disease, and primarily with reference to the production of disorders of the nose, throat, larynx, and voice. Among the problems of the author's own clinical experience which have forced themselves on his attention are such as:—

(1) Why is it that after surgical removal of all obstructions in the nose and nasopharynx mouth breathing often persists, and obstinately defies correction?

(2) Why do so many persons with no apparent disease of the nose, throat, or larynx, or body generally, have recurrent sore throat, huskiness, hoarseness, and fatigue, on slight use of voice?

(3) Why is the posterior segment of the larynx so much more pre-eminently and obstinately involved in most laryngeal affections than

42 Spicer: *Model demonstrating "Back" & "Belly" Breathing*

its other parts? (common bacterial invasions, tubercle, postericoid cancer, &c.)

(4) Why do most people sniff, gasp, pant, or make other unpleasant noises, when using the voice, on slight activity, or even at rest?

Continued investigation of these and many allied problems has thrown the author back after much practical, experimental, clinical, and literary study to the conclusion that the basic factor in causation is the manner in which the respiratory mechanism is used, and whether its activities are favoured by, or opposed to, those of the other muscular mechanisms, and pre-eminently that dealing with body equilibrium or balance—i.e., with attitude or posture of head and trunk, with reference to the unceasing action of the force of gravitation.

If at this point any objection should be made that the principle is already accepted that an explanation is to be found in "something or other wrong with the mode of respiration or production of the voice," he would ask the objector to explain why such a large proportion of persons submitted to "breathing exercises" and "voice training" at the hands of experts and specialists, even under medical sanction and approval, have ended in widespread, signal, and dismal failure.

Is the matter really understood by laryngologists or vocal and respiratory experts, and can they get practical results in the desired direction? If not, why not? Not only the medical profession, but also its clerical, teaching, musical and theatrical sisters, as well as the general body of the public, are clamouring for a satisfactory solution to such questions as are enumerated above.

The terms back-breathing and belly-breathing are used to imply an anatomical and physiological syndrome and embrace the idea not only of the way in which the respiratory and postural mechanisms are habitually used, but also the stereotyped form of the adult individual. In most cases the bony, cartilaginous and ligamentous tissues are not structurally deformed to such an extent that the belly-breather cannot be taught to some degree to adopt back-breathing to his own great advantage. To recapitulate briefly the chief objective marks of each variety:—

Back-breathing is characterized by (1) impulse to fullest spinal extension to start inspiration, straightening out not only dorsal spine (to enlarge chest) but also cervical spine (to enlarge throat) and lumbar spine (to make room for viscera backwards); (2) passive, loose balance of head on spine, no active muscular tension or rigidity; (3) thoracic cage raised, but only by action of the vertebro-thoracic muscles; (4) no

descent of diaphragm or thoracic or abdominal viscera, but base of thorax expanded, on the in-breath; (5) anterior abdominal wall made taut by vertebro-thoracic muscular movement only; (6) on expiration no collapse of spine or chest, gentle but distinct contraction of anterior abdominal walls as action of inspiratory muscles wane, and vice versa on inspiration.

Belly-breathing is characterized by (1) spine collapsed or bent forward, not extended but flexed (round or hollow back) or tendency thereto; (2) chest collapsed, sunken, and contracted; (3) head dropped backward or forward; (4) chest enlarged vertically by vigorous descent of diaphragm (viscera also descend), no expansion, but rather contraction of base of thorax on the in-breath; (5) passively dilated, protruded, or loose anterior abdominal walls; (6) expiration, an uncontrolled elastic recoil, or what are known as muscles of forced expiration habitually brought into play.

The results as seen in the nose, throat, larynx, neck, and voice, are:—

Back-breathing: (1) On palpation of the neck on the in-breath there is a looseness, softness, and free mobility of the windpipe, larynx, and deep structures of the neck, when these are grasped and gently moved from side to side. (2) On each in-breath no descent of the larynx or dragging down of the windpipe is to be either seen or felt. (3) The in-breath is perfectly silent to the ear of the observer; there is no sniff, gasp, or other noise. (4) On auscultation with a stethoscope over the larynx and windpipe, the breath-sounds are of a soft, blowing character, and run into each other, indicating a prolonged easy passage of both in- and out-breaths through the glottis at a low pressure and the absence of any forcible impaction or vibration of the throat and nose structures. (5) Observations of manometric pressure in the nose, when the nasal passages are not obstructed, show respiratory fluctuations of pressure, usually — 5 mm. water on inspiration and + 4 on expiration in back breathing. (6) In absence of nasal obstruction the subject experiences perfect ease, comfort, and absence of effort both in breathing and speaking. (7) The *alæ nasi* move rhythmically, expanding with the in-breath and contracting with the out-breath, but there is no collapse of nostrils. (8) The singing voice is under good control; the tone open, bright, and ringing; trueness of pitch is easily maintained; the *messa di voce* and *portamento*, flexibility and modulation are easily practicable and the voice carries with ease—even the softest tones being heard in a very large hall.

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Belly-breathing: (1) On palpation of the neck on the in-breath, the windpipe and cricoid are felt to be tight and rigid, it is difficult to move them from side to side—evidence of increased strain and pull—and that whether the head hangs forward or is retracted. (2) On each in-breath the anterior cricoid arch of the larynx can be felt and often seen to descend towards the manubrium, tugged down by the windpipe. (3) The in-breath is accompanied by an audible, sometimes noisy, nasal sniff, or a pharyngeal gasp or sucking sound. (4) On auscultation with a stethoscope over the larynx and windpipe the breath-sounds are of a loud, harsh, tubular character, with a well-marked interval of time between them. (5) Similar observations in same case show respiratory oscillations usually about — 25 mm. water (inspiration), + 20 mm. (expiration) in belly-breathing. The increased degree of negative pressure (necessitating suction or cupping of the mucous membrane) must cause chronic congestion, irritation and (if persistent) thickening of the membrane to a commensurate degree. (6) Even when there is no nasal obstruction there is a sense of effort, breathlessness and panting, often when in complete repose and in voice use. (7) The alæ nasi collapse on the in-breath and are blown apart on the out-breath. (8) The singing voice is less under control; the quality tends to be throaty, dull and muffled; the pitch tends to sharpen or flatten, *messa di voce* and *portamento* are difficult or impossible, and voice does not carry well to a distance. If power is used the impression of brute force and coarseness is given, and a bellowing character is manifested akin to the street coal-hawker's cry of "coals."

His investigations on the variations of stress, strain, and friction in the throat, in back- and belly-breathing respectively, led him to discover that in belly-breathing there was a respiratory rotation of the cricoid on the thyroid in an inverse direction to the phonatory rotation which was previously known—an interesting new anatomical and physiological point. This feature is clearly taken advantage of in man to diminish to some extent the ill-effects of belly-breathing in the throat structures. The point was well illustrated in the model.

Not only in this respiratory rotation of the cricoid on the thyroid has Nature shown ingenuity in minimising stresses, strains, and frictions between the adjacent parts of the human machine. For here in the throat, whereas the spinal column is flexible, all angles rounded off, and made as smooth and even as possible by the anterior common ligament and superjacent cushion of muscles, all the parts of the larynx are freely movable on each other, and padded with muscles, &c., as far as possible

on the vertebral aspect ; there is, moreover, a provision for advantageous mutual adjustment of parts through reflex nervous action and co-ordination. Similarly all the hard angles and ridges of bone and cartilages in the larynx tend to be rounded and bevelled, and their surfaces and the opposed spinal tissues (in addition to muscular padding) covered with a smooth mucous membrane freely and incessantly lubricated. All these advantages are only too often frustrated by man's ignorance, neglect, or refusal to use his body to maximum mechanical efficiency. The author's present study of the subject is in no sense whatever an examination or explanation of any individual system, but an attempt to determine the general scientific bases which should underlie all sound vocal and physical culture work as well as the best way to apply any scientific findings in the treatment of disease.

To avoid misapprehension it is well to add :—

(1) No deduction has ever been made, or is made, from the working of the model, and no theory is based thereon.

(2) The parts of the model are not constructed to scale.

(3) It is not suggested that the model represents differential quantitative effects.

(4) The model was not constructed with view of "proving" anything, but as "illustrating" a generalization based on a long series of observations on many living subjects over many years.

As the sole ultimate object is scientific truth, discussion and criticism are invited on the fundamental conceptions which the model has been constructed to render concrete and easily intelligible. *See* author's previous papers.¹

¹ *Brit. Med. Journ.*, 1909, ii, pp. 673, 1149.

THE MODEL.

I.—BACK-BREATHING.

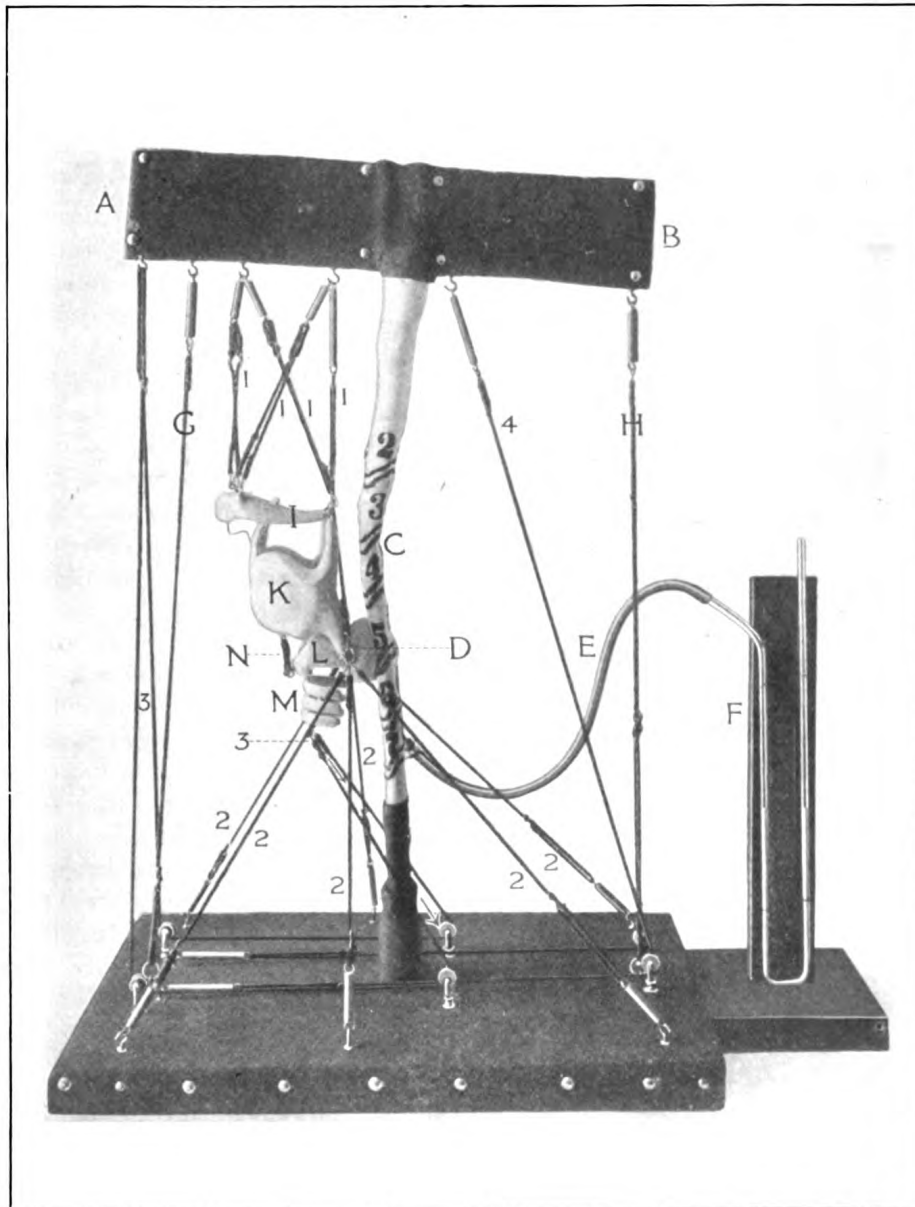
On gently depressing point A on model, it is seen that:

- (1) There is no downward movement of larynx or trachea.
- (2) There is no rotation of the cricoid on the thyroid.
- (3) The strain in the whole air-tube is lessened, indicated by: (a) a shortening of the springs suspending the larynx; (b) the rise of the mercury in the proximal arm of the U tube connected with the air-ball.
- (4) The bezel of the cricoid tends to move away from the spinal column.

II.—BELLY-BREATHING.

By gently depressing point B by a mechanical arrangement, traction is made on the trachea, and the cricoid to which it is attached, in a direction approximating to that in which the inspiration descent of the diaphragm would pull it down. These movements are simultaneous, as in nature. It is then seen that:

- (1) There is a vigorous downward movement of the trachea and cricoid.
- (2) The anterior arch of the cricoid rotates downwards upon the transverse crico-thyroid axis leaving the thyroid.
- (3) All the tensions in the air-tubes are increased, indicated by: (a) the suspension springs being stretched tightly and elongated; (b) the mercury rising in the distal arm of the U tube.
- (4) The heel of the bezel of the cricoid presses against the spinal column with an intensity proportional to the vigour of the diaphragmatic pull and a corresponding compression of the soft tissues between the bosses of the posterior cricoid arch and the deep resistance ridges of the spinal column. This, if continued indefinitely, according to the author's conception, may cause irritation and the requisite abnormal stimulus to cell proliferation, and also to penetration, characteristic of cancer. If the other hard parts be considered, and compared similarly, it would appear that similar augmentation of the force with which these hard parts normally drive and plough into the soft tissues or rub, or squeeze, or press, or stretch them, can be inferred.



A B, horizontal bar representing base of skull; **C**, vertical spring representing vertebral column; **D E F**, air-bag, tube, and pressure-gauge; **G H**, stays to keep apparatus firm; **I I I I**, hyoid; **L**, cricoid; **K**, thyroid; **M**, trachea; **N**, crico-thyroid ligament; **1 1 1 1**, suspensors of throat from base of skull and jaws; **2 2 2 2**, stays attached to ends of transverse axis of crico-thyroid rotation; **3 3**, cords moving the trachea and cricoid downwards in a direction approximating to that in which they would be pulled by the descent of the diaphragm in belly-breathing; **4**, cords producing movement of head backwards such as muscles at back of neck would produce. When **3** and **4** are put to work simultaneously by depressing **B**, an active rotation of the cricoid on thyroid is seen.

DISCUSSION.

Dr. H. J. DAVIS said he did not see how the movement of the vertebræ in the neck could be affected by what was described.

Dr. HARRY CAMPBELL said it seemed to him that in ordinary breathing the larynx as a whole did not move, whether the breathing was abdominal or thoracic; however, the larynx, so far as he could tell on his own person, remained stationary. In extraordinary breathing the larynx descended, but so long as the quantity of air inspired was the same, the effect on the larynx seemed to be the same whether the breathing was abdominal or thoracic. With this proviso, he did not find that the cricoid cartilage moved down any further with deep abdominal breathing than with costal. But with a complete costal breath one took more air into the chest than with a complete abdominal breath, and consequently the larynx actually moved down further in the former case. With regard to the comparative effects of costal and abdominal breathing on the circulation, as set forth by Dr. Spicer in one of his papers, he was opposed to that gentleman's view. Dr. Spicer contended that abdominal breathing made for a sluggish circulation in the splanchnic area, but his own opinion was opposed to that. On taking a deep abdominal breath not only did one enlarge the thorax and so suck blood into the chest by increasing thoracic suction, but by a force-pump action one squeezed the splanchnic blood into the right auricle. An ordinary diaphragmatic breath, therefore, at one and the same time increased the suction action of the thorax and squeezed the blood out of the abdomen into the heart. As regards Dr. Spicer's model, it seemed dangerous to argue from it to the living organism, because it must be remembered that the lungs, owing to the presence of the pleuræ, were able to move up and down within the chest.

Mr. CRESSWELL BABER asked how Dr. Spicer accounted for the sniffing in abdominal breathing more than in chest-breathing.

Mr. HORSFORD said he wished to emphasize the importance of that variety of breathing known as the lower costal as against the abdominal type, and that was the type of breathing which was most successful in developing the voice and in the avoidance of laryngeal strain, which was so common. Laryngologists were brought so much into contact with those who use their voices professionally that it behoved them to study the effects of breathing on the health generally and on the respiratory organs in particular. He agreed with the general opinion now entertained, that costal breathing was one of the most suitable for the effective use of the voice. The great advantage of costal breathing was that it increased the elasticity of the chest-wall, and that was of essential importance in developing the muscles which had to do with the breathing mechanism, in addition to the fact that the enlargement of the thorax

diminished the vocal strain by increasing its value as a resonator, and by acting as a drain to the congested circulation above. He had found that the people who habitually used abdominal methods of breathing were those who more frequently suffered from various effects of laryngeal strain.

Dr. HALLS DALLY desired to say a word on the general position. He would speak on the four questions asked by Dr. Spicer. In answer to the first question, mouth-breathers had used that method for years, and it had become automatic; the practice was very general in children. Though it was wrong, it was of such long standing that it could not be corrected all at once. The nasal passages exhibited changes similar to those seen in other organs which were unused, and in those cases the mouth-breathing was very difficult to correct. In answer to the second question concerning huskiness, &c., he thought that was due to the fact that improper use was made of the voice, by calling into play incorrect neuro-muscular mechanisms for the performance of that object. The reply to the third question he preferred to leave to laryngologists. The transverse axis of rotation of the crico-thyroid cartilage was correct, but it was difficult to prove, because on using X-rays the rays penetrated much of the cartilaginous tissue, and one could not see the rotation except of the cricoid itself. In answer to the fourth question, he thought it was simply because people did not understand the law of atmospheric pressure and had not been taught the principles of breath control. When such people were asked to take a deep breath they did it with great effort, throwing their head back and making a sniffing noise. It was not his experience that during rest most people sniffed, or made unpleasant noises when awake. As far as Dr. Spicer went with regard to the fundamental defect, he was in agreement with him. But that incorrect breathing in itself is not solely responsible for the errors quoted he hoped to show in a forthcoming paper. If Dr. Spicer's model was not "constructed with a view of proving anything," he did not see the utility of it. The cords intended to represent the diaphragm, he thought, pulled in the wrong way. The action of the diaphragm in descent is downwards and forwards, but the cords of the model pulled the larynx and the cricoid backwards, and the vertebral column in the model was pushed forward and impinged on the larynx. He did not think those things took place in actual life. He thought it would be agreed that "belly" breathing was wrong, and the result of it was seen in enteroptosis. He protested against so-called "back" breathing being regarded as the ideal, as it represented only a part of the whole. From repeated and extensive observations Dr. Halls Dally found in every case that initial descent of the diaphragm occurred to greater or less extent preceding the second part of its action in elevating the lower ribs.

The PRESIDENT asked whether Dr. Halls Dally's investigations were made with the abdominal organs *in situ* by Röntgen picture, or whether they were anatomical, when the abdomen was emptied of its contents. The Section was indebted to Dr. Halls Dally for his remarks, and members knew of the important paper which he had read before the Royal Society.

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Dr. HALLS DALLY, replying to the President, said that his experimental observations with the orthodiagraph were made on some hundreds of healthy adults of both sexes during life.

Dr. JOBSON HORNE said it would be generally conceded that the more one saw of the average voice-trainer, the more one sympathized with the average pupil. He had never yet met a voice-trainer who had not told him how many voices were ruined by other voice-trainers. If Dr. Spicer's model were put in front of the pupil to show him how to breathe, he would either give up the task in despair, or would soon be in the hands of a laryngologist. He knew that Dr. Spicer approached the matter in a scientific spirit. In regard to question 3, was Dr. Spicer going to suggest to the Section that by adopting his method, or modifying the way of breathing, that would be the means of preventing the posterior segment of the larynx being so frequently the part involved in tuberculosis? The merest tyro in pathology could not accept such a suggestion.

Dr. H. J. DAVIS said that if one put a man or a boy flat on his back on a couch and asked him to breathe naturally, he would breathe with the abdomen; a woman or girl would breathe more with the chest. The same applied to children of both sexes: it had much to do with sex. It must be assumed that if people lay flat on their backs the spine was straight.

Dr. DONELAN suggested that the subject might be brought forward as a formal discussion at an early opportunity.

The PRESIDENT said Dr. Donelan's suggestion was a matter for the Council; he personally was in favour of such a discussion. He did not think that Dr. Spicer had helped his advocacy by bringing forward the model. Dr. Spicer's reasons were much more convincing than his model, as a model could be made to do anything. All would not accept the term "back-breathing"; "lateral costal" was a better one, and lateral costal breathing could not take place without the action of the diaphragm. It was proved many years ago by Duchenne (quoted in "Brücke's Physiology") that as long as the abdominal organs were *in situ* and kept braced up, the contraction of the diaphragm elevated the ribs. If the liver and other abdominal organs were taken away and the diaphragm stimulated, that did not occur. He (Dr. Grant) thought it was possible to "sniff" well with lateral expansion. But lateral expansion did not seem equal to the requirements of laughing, for which the abdominal muscles were necessary. With regard to the idea that it was erroneous breathing and the rubbing of the cricoid against the spine that caused carcinoma so frequently in that region, he thought it was rather the result of swallowing badly-chewed food; defective dentition was, in his experience, a companion of it. As to the reason why people did not breathe properly after they had had their noses cleared, it would be remembered what Carlyle said as to the characteristics of humanity in general. The Latin poet had well said we knew what was right and did what was wrong. The cause was inability to apply intelligently instruction in breathing.

Dr. SCANES SPICER, in reply, said that he was especially pleased to hear Dr. Harry Campbell and Dr. Halls Dally as experts on the modern physiology of respiration. He hoped their presence signified that they considered some of the propositions the speaker had published, and now again advanced, though calling in question certain accepted physiological teachings, were not rash speculations, but were at all events deserving of discussion and criticism. Dr. Harry Campbell was, without doubt, the father of the scientific application of respiratory mechanics to the treatment of disease, and the speaker was infinitely indebted to him for much light in his works on respiratory exercises, pulmonary physics, and other philosophical dissertations. He fully agreed with Dr. Campbell that on expiration the abdominal muscles should contract and act as a force-pump for the circulation. He also agreed that if the diaphragm descended on inspiration there was increased abdominal pressure, and so in some degree portal drainage was favoured; but he regarded the disadvantages of such descent as overwhelming. If, on the other hand, the great vertebro-thoracic and diaphragmatic mechanism produced thoracic suction on inspiration (acting as a suction-pump for the venous circulation), the portal and splanchnic areas and venous and lymphatic systems were evacuated during both phases and the circulation continuously promoted. Hence he believed that the normal influence of the diaphragm on the circulation was far greater than had as yet, to his knowledge, been suspected. From his preceding remarks it would be seen that he considered diaphragmatic descent as a habit in breathing had a comparatively feeble effect in enlarging the thorax or promoting the circulation, while it caused visceroptosis and many other abdominal, pelvic, and lower-limb diseases, in addition to the commoner nose and throat affections. It was, he believed, a main factor in modern physical degeneration. From his own screen-work over many years he was well aware that this descent was a fact in practically every modern individual who had had no training in the matter; but he could not regard it as "normal," and believed its habitual continuance as the regular mode of breathing must be abandoned in the interest of the health of the public and the future welfare of the human race. He recommended all interested in various aspects of the subject to read Dr. Halls Dally's paper on the "Diaphragm" in the *St. Bartholomew's Hospital Reports*, 1908, also his communications on "Respiration" to the *Proceedings of the Royal Society*, 1909, and *Journal of Anatomy and Physiology*, 1908. Mr. Cresswell Baber had asked him how he accounted for collapse of the alæ and sniffing in belly-breathing. His present view was that the collapsed chest prevented a great part of the lung from expanding, and in order to approximately supply the respiratory need in getting the air down to the expanding part of the lung, the diaphragm made a rapid and vigorous descent, sucking the air out of the main naso-bronchial passages and causing a great fall of air-pressure as compared with the external atmosphere. The alæ therefore fell in, and the vibration of their edges by the inrush of fresh air caused the sniffing sound, while a similar vibration of the other movable parts of the air-tract—e.g., palate, epiglottis, cords, and ventricular bands—caused those other sounds

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known as "heavy" breathing. He thanked Dr. Horsford, Dr. Davis, and Dr. Donelan for their apropos remarks, especially the latter for his suggestion that the Section should hold a discussion on the subject. He thought the Section might do worse, because it was especially important that vocal people could depend on using their vocal and respiratory mechanism at all times, and so much of the work of the members of this Section was concerned with theatrical, musical, and other professional voice-users. What he had advocated, if thoroughly carried out, would do much to remove altogether their recurrent voice troubles. He knew from experience that this change of posture and method of breathing from belly-breathing to back-breathing was one of the finest weapons in the specialty, and if he could get members to investigate and think over the subject by his communication that day he had done well. He did not grasp Dr. Jobson Horne's point, but he would repeat his previous statement, that in belly-breathing the posterior cricoid arch was liable to abnormal friction, compression and strain, causing irritation, congestion, and swelling—often extreme—of the mucosa or posterior wall of larynx and adjacent part of the trachea. This was, therefore, mechanically and biologically a favourable nidus for the lodgment of the tubercle bacillus, if such were present in the sputum, and it appeared to the clinician a pre-eminently favourable one from the comparative frequency of tubercular lesions in that site.

Laryngological Section.

January 7, 1910.

Dr. J. DUNDAS GRANT, President of the Section, in the Chair.

A Case of Extrinsic Carcinoma of the Larynx.

By G. SECCOMBE HETT, F.R.C.S.

THE patient, a man aged 65, was admitted to University College Hospital on June 7, 1909, having had difficulty of swallowing of six weeks' duration. On laryngeal examination by Mr. Tilley, a large, rounded, greyish-white mass was found immediately behind the free portion of the epiglottis, and completely filling the entrance to the larynx. Further, Mr. Tilley found that the larynx proper was free from disease, and that the growth probably sprang from the region of the aryepiglottic fold or the base of the epiglottis. The whole appearance was that of an epithelioma. There were some enlarged glands in the neck.

On June 14, 1909, operation by Mr. Wilfred Trotter: Laryngotomy; dissection of the anterior and posterior triangles on the right side; longitudinal pharyngotomy after resection of ala of thyroid and cornu of hyoid; excision of growth; suture of mucous membrane.

The patient and the growth removed by operation were shown.

DISCUSSION.

Dr. W. HILL said Mr. Trotter was to be congratulated on the happy result of the case, especially as in his (the speaker's) opinion the chances were so much against it being successful. It was an extensive pedunculated growth, springing from a moderate base, and had been taken away without removing

much of the larynx; he understood that the arytaenoid only had been removed and some mucous membrane in the neighbourhood. He was surprised that, under the circumstances, no recurrence had taken place within six months. If many such cases were recorded, it would of course be necessary to reconsider our attitude concerning partial laryngectomies. The tendency in recent years had been to regard a total laryngectomy as the only reliable procedure in operable cases of carcinoma of the laryngo-pharyngeal party-wall.

Dr. SCANES SPICER said the size of the tumour-mass in the present case was the largest he had seen removed by laryngotomy; in this case he was much struck by the fact that the most advanced spot of degeneration was round about the muscular process of the arytaenoid. Mr. Trotter appeared to have removed the whole of the disease, and he (Dr. Spicer) thought the prognosis was very favourable from the extraordinarily quiet and healthy state of the larynx and pharynx on laryngoscopy.

Dr. FITZGERALD POWELL thought that the thanks of the members of the Section were due to Mr. Trotter and Mr. Hett for bringing forward this interesting case. It was to be noted that this operation was lateral pharyngotomy with partial laryngectomy. He presumed that the growth was in the pharynx; possibly there may have been difficulty in locating its origin. He should like to hear the opinion of the operator as to the necessity for the preliminary laryngotomy, and if it were not possible to do the operation without the laryngotomy. Anyhow, this operation of lateral pharyngotomy seemed to get very good results, as in this case, and he congratulated Mr. Trotter on the present condition of the patient.

Mr. HERBERT TILLEY said the case came into his department first, and the size of the growth was extraordinary, being level with the top of the epiglottis. It was impossible to see the larynx at all. He could only tell where the growth originated by examining it with his finger, when he found it grew from the upper margin of the larynx, on the right side. Its nature seemed quite obvious. There was an enlarged gland in the neck, which he supposed was one of the reasons which prompted Mr. Trotter to operate on that side and deal with the gland at the same time. In spite of the size of the growth, the man could swallow his food and breathe without apparent discomfort.

Dr. STCLAIR THOMSON asked whether it would be possible to have sections of both the tumour and the gland, because, while agreeing as to the remarkable success of the case, he thought Mr. Trotter had had some luck about it. The piece exhibited showed the most distinct pedicle he had ever seen in any malignant growth, and it seemed not to have infiltrated the larynx at all. The piece removed looked as if it had a comparatively narrow pedicle, which was fixed to the back of the arytaenoid, and the lack of infiltration was shown by the fact that the cord and arytaenoid on that side still moved. There were cases of malignant disease of the larynx—endolaryngeal cases—which had been pedunculated and had been removed by endolaryngeal methods, which that Section

generally did not approve of, and yet remained free from recurrence. There was the greatest variety in malignant growths, but he thought cases which were so distinctly pedunculated almost bordered on the innocent; and if the microscope showed anything unusual in these cases, it would be very useful.

Mr. TROTTER, in reply, demonstrated the specimen, showing that, while one part of the growth was actually pedunculated, the remainder was a typical epitheliomatous ulcer. There could be no doubt about its nature. It was examined microscopically, and he would be glad to provide the Section with a slide. Quite half a dozen glands were found distinctly enlarged and invaded by the growth. He wished to show the case because of the nature of the operation which had been performed. He did not share the view that in most of these cases total laryngectomy was necessary; and he thought the idea that it was usually necessary was one of the reasons why operation was so often delayed. With regard to the danger of recurrence in this case, he had been able to use this method of local excision without laryngectomy in two other cases—one operated on thirteen months ago, and one eight months ago: both patients were free from recurrence. The principle which should be adopted in the treatment of carcinoma of the upper opening of the larynx was the same as with malignant growths elsewhere—viz., that the tumour should be in the centre of the tissue removed. The method of doing a set formal operation for malignant disease—such as removing the tongue for cancer of that organ, or removing the larynx for cancer of the upper opening—was in his view not based on a satisfactory principle, and led to delay through dread of the mutilation involved without offering any compensating security from recurrence. The only other principle of treatment on which he desired to lay stress was that the growth should be attacked from the mucous surface. When doing total laryngectomy one began the removal of the growth without sufficiently exact knowledge of its extent. Every operation should begin with a stage which exposed fully the free surface of the tumour. The growth could then be disinfected with the cautery, and could be cut out with due regard to its extent; if that was not done, one was liable to cut into the growth during removal. He thought the essential feature of a lateral pharyngotomy for removing tumours of the upper opening was the removal of the posterior part of the ala of the thyroid cartilage and the great cornu of the hyoid, by which procedure one got access to the whole of the laryngeal part of the pharynx. Then by dividing the constrictors one came down on to that part of the pharynx in which the mucous membrane was so lax that, as a rule, the tumour could be cut out and the part stitched up again, so that the patient had no trouble with swallowing afterwards. In the case of the patient shown there had been rapid and complete recovery of normal deglutition, and there had been no leakage from the pharynx into the neck wound. This had been of great advantage as, on account of the extensive involvement of the glands, the wound in the neck was very large.

Further Notes on a Case of Hoarseness in a Woman aged 60.

By G. C. CATHCART, M.D.

THIS case was shown at the last meeting.¹ It was then impossible to see the cause, owing to the great swelling of all parts of the larynx. The swelling has now disappeared and the voice is natural, the cords being easily seen.

DISCUSSION.

Dr. WATSON WILLIAMS said he would be glad to know what treatment was adopted. He remembered the case at the last meeting; there was a great deal of glandular thickening. He believed he suggested for it the name of glandular laryngitis.

The PRESIDENT (Dr. Dundas Grant) said the extent to which the recession of the ventricular bands permitted improvement in the voice was worthy of note.

Dr. CATHCART replied that the treatment consisted simply of inhalation of compound tincture of benzoin.

Complete Occlusion of Left Nostril by Deflected Triangular Cartilage in a Girl aged 7½.

By G. C. CATHCART, M.D.

THIS patient was sent by her medical man to have the tonsils and adenoids removed. Two very large tonsils and a large mass of adenoids were removed, but so far as the obstruction to breathing is concerned she is no better. She still snores loudly at night and sleeps with open mouth. The case is shown to elicit opinions from members as to the advisability of performing a submucous resection in so young a child.

DISCUSSION.

Mr. STUART-LOW said that he would certainly operate on this case, because the obstruction was so great, and because the child was suffering pain which increased whenever she blew or touched the nose. The pain was due to a spur having developed upon the most prominent portion of the deviation, which impinged against the outer wall of the nasal cavity. A good deal of doubt

¹ *Proceedings*, p. 31.

was entertained among specialists as to the advisability of performing a resection in such young subjects. There was certainly a danger of a falling in of the lateral cartilages if too extensive an operation were performed in many of these cases. Mr. Stuart-Low was strongly in favour of a modified operation being undertaken, but one had to be careful not to remove too much of the supporting cartilage of the septum. The soft structures should not be disturbed more than in the least degree possible, and union by first intention should be obtained. This could be best accomplished by inserting a layer of oiled silk next the wound before packing the nostril.

Dr. PEGLER said he advocated operating upon these cases in children, and could give very good reports of those he had treated from the age of 5 years upwards. His method consisted in simply slicing off the projecting parts of the cartilage with a fine knife, and liberating the septum above and below with his fissure forceps. When displaced to the required degree, the septum was kept in place by means of a suitable splint. He preferred this plan to submucous resection in children. It was quickly done and gave an excellent result.

Dr. H. J. DAVIS asked if any members had seen cases of falling in of the bridge of the nose after any operation on the septum. There seemed to be such an idea in the profession. Recently he had seen two practitioners who stated they could not advise patients to have the operation done, because of the danger of falling in of the bridge. He had seen cases with a septal perforation, but he had never yet seen one with a sunken bridge, the result of removal of the cartilage.

Dr. SCANES SPICER said he believed the idea of a falling in of the bridge after submucous resection was largely theoretical. A similar objection was raised in this Society when he brought forward some antrum cases after his original radical operation, now known as the Caldwell-Luc operation, in which it was said the face would fall in. He knew the critic had never seen such a thing at the time the statement was made. In his experience of submucous resection of nasal septum he had had no case of falling in, but he had seen a very slight dimpling of the bridge more than once. He had operated on three cases between the ages of 5 and 6 years, and, when seen three to four years afterwards, they had shown no deformity. There had been no sign of the operation from the outside. He did not think Dr. Pegler's method of removing a lot of tissue was so good as a strict submucous resection; and it should be done at once, as the development of this child's face was being affected. The obstruction to the passage of air on one side was producing asymmetrical development of the face and orbit, and there would be correlated deformity of the eyeball leading to refraction errors, astigmatism, and squint. This child already showed an alternating internal cast.

Dr. FITZGERALD POWELL said that in his opinion it was very unwise to operate by Killian's method on children of such an age. He could not see any good to be gained by doing so, unless in cases of deafness or ear complications,

when it might be considered. One should wait until the fifteenth or sixteenth year, when the nose had reached a fuller development and the danger of disfigurement was almost *nil*. He had never seen strabismus caused by nasal obstruction.

Dr. LAMBERT LACK said he had seen very considerable falling in of the nose, and there was an undoubted danger in that respect. There did not seem to be any danger in adults, and in children the danger was remote rather than immediate. The deformity was often first noticed six months, or even two years, later. The operation seemed to interfere with the growth of the nose, and the bridge did not consequently reach its normal development.

Dr. DAN MCKENZIE said it was difficult to settle the question with regard to the occurrence of deformity which might result from operating upon the septum of young children. The submucous resection had not been generally practised long enough to enable a decision to be arrived at; in other words, we must wait until the children who have been operated upon grew up before we could be sure whether or not deformity was a danger. Certainly, the removal of growing cartilage would seem, *a priori*, to tend to stunt the development of the septum.

Mr. HERBERT TILLEY said that in somewhere about his fifteenth case at Golden Square he put up his Killian's sharp knife too far, and there had always been a small dimple between the nasal bone and the part below. It was a young adult, and the only case of the kind in which he knew that he had made this mistake.

The PRESIDENT said his feeling was that the obstruction was so great that a carefully-carried-out, discreet submucous resection was justifiable, and would give the best results in this case. If a sufficient amount were removed by simple sawing, there would be perforation.

Dr. CATHCART replied that the case was peculiar on account of the age. He had seen one case of distinct falling in of the bridge, with the result that the nose looked like a congenital syphilitic one. It would be of no use to do a window operation, as the front of the cartilage projected into the left nostril, and unless the front edge were taken off, the obstruction in front would not be removed.

Tuberculosis of Pharynx.

By ANDREW WYLIE, M.D.

THE patient was a clerk, aged 32. About three months ago he noticed a small spot on the right tonsil, which has gradually increased. For several weeks there was pain on swallowing. History of fistula six years ago. Severe cough and spit in the mornings; slight night

sweating; weight reduced by $1\frac{1}{2}$ st. within the last few months. No family history of tuberculous disease; no history of specific disease. Four weeks ago the left ear began to discharge, commencing without pain.

Upon examination, the right anterior pillar of fauces is ulcerated, and on the posterior pillar is an ulcerated patch. On drawing forward the right anterior pillar of the fauces a large excavation is seen. Dr. Wyatt Wingrave found tubercle bacilli in great numbers on examination of the pus from the diseased surface, and also in the sputum. Epiglottis somewhat enlarged and arytaenoid cartilages swollen, but no ulceration or infiltration of the vocal cords. Crepitant râles at both apices.

DISCUSSION.

Mr. HETT asked whether Dr. Wylie regarded the case as tuberculosis of the tonsil. There was a large hole between the pillars of the fauces, and Dr. Wylie had said that the ulcer started on the tonsil. He had seen about six cases of tuberculosis of the tonsil starting as an ulcer, which gradually ate away the tonsil and left a hollow between the pillars.

Dr. STCLAIR THOMSON said the epiglottis was not very large now, and the arytaenoids looked normal. Scrapings would show what it was, and it would be interesting to learn the further progress of the case. He was surprised that Mr. Hett had seen six cases of tuberculosis of the pharynx, as he (Dr. Thomson) looked upon it as one of the rarer diseases, though lupus of the pharynx—a different condition—was known to them. The cases he had seen had been very acute infections. Even if the lungs had not been much affected, the patients had exhibited a high temperature and bacilli were found in clumps. There had also been exaggerated pain, and he had not known such a patient live six months. He agreed that, clinically, this case was tuberculosis, but it was mild compared with the cases he had seen.

Dr. SCANES SPICER said there was a neoplasm attached to the back of the anterior pillar of the fauces on that side, of the size of a black currant, and he asked what that was. Also, what treatment had been carried out? He thought something active should be applied locally, such as galvano-cautery or lactic acid.

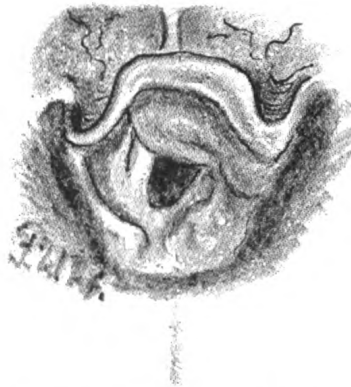
Dr. JOBSON HORNE said he was about to observe, as Dr. StClair Thomson had done, that Mr. Hett was fortunate in having seen six cases of tuberculosis of the pharynx in so short a time. Some years ago he (Dr. Horne) microscoped a large number of tonsils removed from subjects who were suffering from tuberculosis of the cervical lymphatic glands, and he had the greatest difficulty in finding tubercle bacilli in the tonsils.

Dr. FITZGERALD POWELL said he thought it was not very uncommon to see tuberculosis of the pharynx; he had himself seen several cases. And he did not think it should go out from the Section that because a patient had that disease in the pharynx he would not live more than six months. He had seen cases in which the local lesion got well after treatment, and the patient lived for a considerable time.

Case of Laryngeal New Growth for Diagnosis.

By P. WATSON WILLIAMS, M.D.

E. H., AGED 65, came to the Bristol Royal Infirmary on July 7, 1908, complaining of cough, expectoration of phlegm, and hoarseness, since Christmas, 1907. The fauces were a little congested, but



Laryngoscopic appearance of the growth.

examination of the larynx revealed a pink, irregular, nodulated neoplasm involving the left arytenoid eminence, the left ventricular band of the posterior surface of the left side of the epiglottis, and extending to the glosso-epiglottic fold, and projecting over the glottis so as to completely conceal the left vocal cord, except that the posterior one-third was visible when looked at obliquely from the right side, when it was seen that both cords moved freely on phonation. Two small, hard, movable glands were felt behind and above the angle of the jaw on the left side of the neck. It was diagnosed as epithelioma, and on account of the patient's age it was felt inadvisable to operate on a growth which had

already involved the glosso-epiglottic fold and was complicated by enlarged glands.

The patient has been seen several times since. At first the growth gradually increased in size, yet without obviously extending to the regions not involved at the first inspection. Now, in December, 1909—that is, two years after the growth was first causing hoarseness, and seventeen months after he was first seen—the condition remains apparently much the same. The patient's general health does not appear to have deteriorated meanwhile.

The possibility of its being a tuberculous growth was considered, but a Pirquet's reaction was negative, and there are no general symptoms or lung conditions to support this view.

When first seen the patient was put on 60 gr. of iodide of potassium daily for some months.

DISCUSSION.

The PRESIDENT said it seemed to be a very slow growing malignant growth.

Dr. W. HILL said he thought the growth was unlikely to be a true epithelioma; it was very possibly an endothelioma. It was not ulcerated, and was very slow growing. He did not regard it as typical carcinoma, because a growth in that lateral position would grow fast and infect the neighbouring glands.

Dr. SCANES SPICER said he did not think that the long duration without change could be used as evidence of the nature of these growths. One such case he had shown for five years before this Society. It was repeatedly removed, and appeared innocent, but ultimately turned out to be malignant. The lobulated tumour in the present case appeared to be encapsuled, and he thought that in the early stages of some of these cases the whole of the proliferating neoplastic tissue was encapsulated by condensation of surrounding tissues. At what point could such a mass be said to pass the border-line? This man was leading a healthy, outdoor life—that of a gardener. If he had been of a sedentary occupation the case might have been different, and a gastro-intestinal autotoxæmia from portal stagnation have speedily ensued. Active life, well-evacuated portal system, and healthy blood and neurons had, perhaps, prevented the local lesion from becoming aggravated, and this might have something to do with the slowness of its progress.

Mr. HERBERT TILLEY thought that if it were possible to look under the lower edge of the swelling some ulceration would be found. If there were any doubt about this, Dr. Williams could prove it by using the direct method and passing a mirror through the tube. If it were ulcerated it would be easy, with punch forceps, to remove a piece, and study the nature of the growth. With

regard to the latency of some malignant growths of the larynx, he had recently to deal with a case which Mr. Butlin, Sir Felix Semon, and himself had been seeing at intervals for seven years. There was a little thickening of one vocal cord, and nobody seemed inclined to commit himself as to its real nature. The patient was an old man, and some months ago, as he began to get more hoarse, the larynx was opened and the growth was found to be typical epithelioma.

Dr. STCLAIR THOMSON said that in the American Laryngological Association's records there was a case which was watched for twelve years,¹ in which the cord presented the appearance of a "field of wheat which had been snowed on." That case passed through several hands in New York, and finally came to operation, when it was found to be a slow-growing intrinsic epithelioma.

The PRESIDENT suggested that the condition of the glands almost clinched the diagnosis.

Dr. WATSON WILLIAMS, in reply, said it appeared from the discussion that there was not much he ought to have done which he had not done. Mr. Tilley's useful suggestion was safeguarded by the remark that he would do it if ulceration were discovered. He (Dr. Watson Williams) had not found any ulceration; and he said that, although there was a great temptation to remove a portion for diagnostic purposes, unless one were prepared to operate it would be bad practice. And if it were malignant he would not operate, as such an extensive operation as alone could be successful would be bad practice at the patient's age. The glands in the neck had slightly enlarged lately, but he did not think the growth in the larynx was any larger than it was eighteen months ago.

A Case of Endothelioma of the Ethmoid.

By CHICHELE NOURSE, F.R.C.S.Ed.

WHEN first seen in October last the patient, a well-nourished woman aged 29, complained of complete obstruction of the right nostril, with yellowish and bloody discharge, but no pain. The nasal septum was deflected to the left, and the right nostril blocked by a smooth red swelling, which seemed to be the much-enlarged inferior turbinal. Eighteen months before, some slight intranasal operation had been performed elsewhere with temporary relief; prior to that the nostril had been stuffy for two years. After an unsuccessful attempt to remove

¹ Harmon Smith, *Trans. Amer. Laryng. Assoc.*, New York, 1909, p. 79.

the growth with a turbinotome, the nostril was partly cleared with forceps. The growth was friable and not very vascular. Specimens were handed to Dr. Wingrave, who reported that it was an endothelioma. The right antrum was absolutely dark on transillumination. In puncturing through the nasal wall no bone was encountered. The antrum contained no fluid.

The operation was performed on December 16. After a preliminary laryngotomy, a flap consisting of the soft parts of the cheek and upper lip was turned outwards as for excision of the superior maxilla. The maxillary antrum was then opened through the anterior wall and the ascending process of the superior maxilla and a part of the nasal bone removed, so as to effect a complete exposure of the right nasal fossa and of the antral cavity. The antrum contained polypi and an irregularly thickened fleshy lining, which was easily detached from the bone, leaving a roughened surface. The bony inner wall had almost disappeared. Parts of the anterior, posterior, and orbital walls were much thinned. There was no bulging. The superior nasal fossa was unaffected. A pedunculated mass, the size of a small walnut (weighing 18 grm. after removal), projected into the middle fossa from the anterior part of the ethmoid; its attachment was surrounded by a cluster of small prominences on the thickened mucous membrane. From this point downwards the whole mucous lining of the outer wall and floor of the right nasal fossa was thick and uneven. The partition between the antrum and the nose consisted, except at its edges, of a fleshy curtain without bone. The whole of the diseased tissue was stripped from the nose and the antral cavity, leaving the bone bare, except where parts of the thin bone came away with the growth. The thin bone of the posterior antral wall gave way, and the fat in the zygomatic fossa was exposed. The nasal duct was divided close to the lachrymal sac, and removed with the tissue in which it was involved. The middle turbinal body, though apparently unaffected, was removed. Chloride-of-zinc solution was applied to the cavity, the antrum was lightly plugged with gauze, and the wound closed with sutures. Healing took place rapidly. There was some œdema of the cheek and eyelid for about a week. The present condition of the patient is satisfactory.

Dr. Wyatt Wingrave's report is as follows: "The growth consists of rods of cells, similar to endothelium, of varying size, separated by a stroma of colloid-looking substance containing a few fixed connective-tissue cells and plasma cells. In the more developed regions the stroma is scanty or absent, endothelium being predominant; while, in the

intermediate stage, alveolation is well marked. Where the cylindrical arrangement prevails there is a distinct lumen filled with colloid substance, having the appearance of thyroid gland, but unlike the latter, not giving the thionin reaction, but selecting acid stain. Many of the



FIG. 1.

Microscopic section of growth under low power (1 in.).

sections show a boundary of dense fibrous tissue, but there is no definite and regular capsule. In parts the bone is involved, with considerable involvement of the periosteum. The individual cells are fusiform or circular according to the axis of section. The nuclei are oval, containing

very small karyocytes. Here and there heteromitoses are seen. Their cytoplasm is clear. The middle turbinal proves to be invaded by new growth. In the antral lining a small amount of lymphocytic infiltration is present, with clusters of plasma cells, and elongation of gland ducts, lined with palisade epithelium; therefore most of the thickening is due to prolonged inflammation."

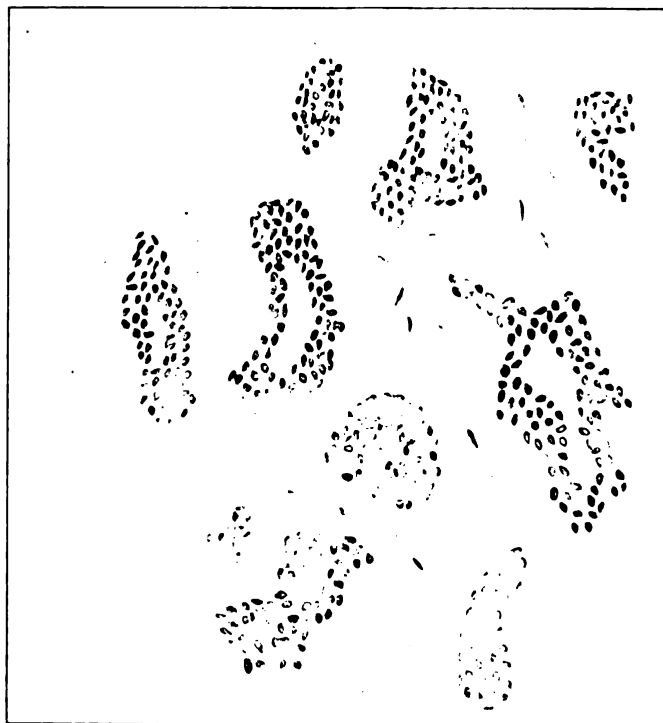


FIG. 2.

Microscopic section of growth under high power ($\frac{1}{8}$ in.).

DISCUSSION.

Mr. STUART-LOW said the result was apparently very satisfactory, as the disease had evidently all been removed, and there appeared to be no likelihood of any recurrence of the symptoms. He had assisted Mr. Nourse in a similar case some time ago, in which the final result was also very good. Mr. Stuart-Low had recently had two cases, also in females, in whom it was of paramount importance not to produce any facial blemish. To avoid this he had performed

a modification of Jensen's method for the radical antral operation. The buccolabial fold was very freely incised from the incisor fossa backwards, and the nasal cartilages and cheek forcibly retracted towards the eye of the same side with powerful retractors. The anterior antral wall was almost entirely removed, including part of the ascending process of the superior maxillary bone. This gave exceedingly good access to the nasal cavity and easily allowed the most remote portions of the tumour—a columnar-celled epithelioma—to be thoroughly cleaned out. The result in both instances had been most gratifying, as not only was the malignant growth eradicated without recurrence, and six months had elapsed since the operation, but there was no mark nor any deformity whatever on the face.

Dr. WATSON WILLIAMS desired to congratulate Mr. Nourse on the complete success of the case; but where malignant disease involved the ethmoidal region one could not speak with certainty until the case had been under observation a considerable time. He would have approached the case by the osteoplastic method, which he adopted in a case of malignant growth involving the septum as well as the ethmoid cells high up. He showed the case before the Section about a year ago, and illustrated the operation.¹ Making an osteoplastic flap on the side of the nose and turning it on a hinge, so as to expose the whole area of the nose on that side, had the advantage of giving very free exposure and leaving no noticeable cicatrices. It was the facial incisions which caused the deformity in Mr. Nourse's operation, but, of course, such deformity was nothing compared with the effectual removal of the growth. In such a case he thought one ought not to attempt intranasal operations.

Dr. PEGLER said the type of endothelioma which was here shown seemed to him to correspond with the growths well known as occurring in the soft palate and also in the parotid gland. This was the first case, so far as he was aware, that had been brought before the Section, which had started from the middle turbinal.

Dr. SCANES SPICER said he had had several cases of antral and ethmoidal tumours which were said to be alveolar sarcomata, and to be malignant, by the histologists. One such case fifteen years ago, who was said to have had malignant sarcoma, was well now, there having been a scraping out of the whole disease through the bucco-antral wall and the nose. He asked whether Mr. Nourse had considered the intranasal and bucco-antral operation in the case. There was a decided scar on the nose, and the patient was aged 29, so that, if possible—and there were no strong contra-indications—he would have preferred to try an internal operation first. We wanted further definition and information from the histological standpoint. Personally, he was not inclined to do a radical external operation simply on the strength of the histologist finding endothelioma or alveolar sarcoma. Many such cases did not recur after thorough removal and permanent drainage, and clinically were not malignant.

¹ *Proc. Roy. Soc. Med.*, 1908, i (Laryngol. Sect.), p. 116.

Dr. JOBSON HORNE agreed with Dr. Scanes Spicer that there was no form of growth which lent itself more readily to two opinions than such a one as the present. As there was not a specimen of such a growth in the cabinet of microscopic specimens from this Section of the Society, it would be a great help to the Section if the specimens from the case which Mr. Nourse had exhibited could be thoroughly examined, and if the Morbid Growths Committee were allowed to cut their own sections.

Mr. HERBERT TILLEY said reference had been made to the question of the advisability of removing a growth of the kind from the outside or from within. The most excellent method of getting a lot of room for the combined maxillary and ethmoidal operation was an extension of Denker's operation for dealing with chronic maxillary suppuration. In order to turn up the soft parts from the anterior antral wall it is necessary that the patient's mouth be closed, and this will render the maintenance of general anæsthesia a difficulty. To obviate this a laryngotomy should be performed and a sponge inserted above the larynx. An incision is then made in the gingivo-labial fold from one malar process to the other, the soft parts pushed upwards, and the ascending process of the superior maxillary bone removed as well as the anterior antral wall. Free access is thus gained to the nasal cavity, and, on replacing the facial tissues, immediate union of the soft parts usually occurs and no external scar is necessary.

Dr. LAMBERT LACK desired to support Mr. Tilley's remarks concerning Rouge's operation, and especially to emphasize the necessity of a preliminary laryngotomy. He had also tried Dr. Watson Williams' operation, and would be inclined to say generally that if the growth came from the floor of the nose, the inferior turbinate, or the outer wall or antrum, he would do Rouge's operation; whilst if it arose from the ethmoidal region, it was better to get direct access by Dr. Watson Williams' method, which left remarkably little scarring.

Dr. STCLAIR THOMSON said he was hoping that Dr. Lack would have mentioned some cases which he showed of malignant growths removed endonasally without there being any recurrence for several years. They were shown at the same time as Dr. Bond's case of an old man, which went on for years with removal of recurrences and glands from the neck. That old man was on hand at Golden Square for some years.¹ (Dr. Lack intimated that recurrence took place in two years.)

Dr. WILLIAM HILL said the only time he had had a nasal endothelioma operated upon by Rouge's method he regretted it. The pathologist reported it to be highly malignant. The whole thing was very successfully removed by a surgical colleague, but it quickly recurred. The patient was very old, and on that account a further operation was refused. He (Dr. Hill) snared and curetted him under an anæsthetic on two occasions—one side on each. The whole condition cleared up, and it had not recurred to any extent. Two years

¹ *Proc. Laryngol. Soc. Lond.*, 1895-6, iii, p. 88, and 1896-7, iv, p. 4.

later he died, when aged 78. In that case Rouge's operation proved to have been quite unnecessary. In cases of endothelioma he did not believe in the microscope as a sure and certain guide to clinical malignancy. Some of them cleared up under "neoformans" injections and under radium, others after removal *per vias naturales* by simple measures. The degree of malignancy in these cases could not be gauged by macroscopic nor by microscopic appearances in his experience.

Dr. DAN MCKENZIE said it was a question whether one should approach the field intranasally, through the upper lip, or through the external parts. The present discussion on the point had not clarified his own ideas very much. A paper had recently appeared by Dr. Price-Brown, who had been treating sarcoma intranasally by means of the galvano-cautery and snare, and apparently with considerable success.

Mr. NOURSE, in reply, said he had considered the various modes of access to the growth, and decided it was situated too high in the nose to be satisfactorily reached by any other route than an external operation. It seemed to him that one of the most interesting parts of the case was the histological character of the tumour. The operation was performed only three weeks ago, and in a few weeks the external scar would be much less visible; there was still some inflammatory exudation in the flap. In answer to Dr. Horne's suggestion, he believed Dr. Wingrave had still a piece of the tumour uncut, and, if so, it was at the service of the Section.

Specific Ulceration of the Lower Lip, Inside of the Cheek, and Tongue.

By CHICHELE NOURSE, F.R.C.S.Ed.

THE patient, a young man, aged 22, was first seen on November 30 last, when he complained of soreness of the mouth and tongue, which had troubled him for about a month. On the inside of the lower lip and on the buccal surfaces of the cheeks were patches of superficial ulceration with whitish raised surfaces, looking like mucous patches. There was also a small ulcer of a similar character on the right border of the tongue. No history of syphilis could then be obtained, nor were any signs of past or present specific disease to be found elsewhere, but now the patient admits that he had a sore eighteen months ago. The ulcers are now smaller, and slightly changed in appearance; they are still very painful.

A scraping from an ulcer yielded no specimens of *Spirochæta pallida*, but *Spirochæta fætida* and *Bacillus fusiformis* were present in numbers. Wassermann's reaction was negative. (Wingrave.)

DISCUSSION.

The PRESIDENT said he thought they were secondary mucous patches on the cheek, approaching leucoplakia in character. There was a history of a venereal sore eighteen months ago, and the patient was a smoker.

Mr. NOURSE, in reply, said that when the man was questioned at the hospital he denied ever having had a sore, and no trace of a scar could be found. Dr. Grant's opinion was confirmed by the result of treatment, because as long as the patient was having simple antiseptic treatment he remained *in statu quo*. Since he had been taking biniodide of mercury the appearance of the lesions was changing and the ulceration had become less.

Bands between Eustachian Tube and Pharynx.

By E. A. PETERS, M.D.

F. G., WOMAN, aged 29. Two bands are to be seen passing from the lip of either Eustachian tube to the pharyngeal wall. The bands are not quite symmetrical, and one is continued as a cicatricial band across the pharynx. Chronic otitis media exists on both sides. The patient does not report scarlet fever or measles. An operation for adenoids was carried out at the Evelina Hospital eighteen years ago. The question is whether the bands are natural or post-operative.

DISCUSSION.

Dr. JOBSON HORNE said the question raised by Dr. Peters was "Are the bands natural or post-operative?" This problem had presented itself to Dr. Horne some years ago; and after examining a large number of post-nasal spaces he had seen many similar cases, but had not satisfied himself that they were traumatic or that they were natural.

The PRESIDENT said he had frequently seen bands in cases which had not been operated upon at all; but in those the bands were rather more succulent in nature, and evidently had been shrivelled-up masses of adenoid tissue. In the present case he thought the very long cord which stretched across was traumatic or post-operative. Probably more such cases would be found if looked for. The deafness in the right ear might be favoured by several

small bands above the right Eustachian tube, and it would be desirable for them to be broken down. But it was also important to find a means of preventing them from re-uniting. He had not himself succeeded in the latter, and would be glad if any other member had.

Dr. SCANES SPICER said he thought that, in this case, where there was a series of bands transversely white, and like ordinary cicatrices, on the left side, they were traumatic. The bands which were left from atrophied adenoids, in his experience, were longitudinal in Rosenmüller's fossa, and were usually red vascular stripes.

Dr. PEGLER said he would advise having the nose thoroughly cleansed and the mucous deposits removed for the benefit of the hearing, and then leave the case alone.

Dr. H. J. DAVIS said he thought that if the bands were post-operative they would consist of scar-tissue. By removing a small piece and examining it microscopically, one could tell at once whether this was so or not.

Dr. STCLAIR THOMSON said he went practically into the question after a discussion before the Laryngological Society some ten years ago, and concluded that the band might be either post-operative or natural. In some cases there was a distinct history of no operation having been performed, whereas in many cases in which adenoids were removed in the middle, and left with granulations along each side, one saw such conditions afterwards. He also observed that a number of cases had no affection of the ear, and there seemed to be no connexion between the two conditions.

Dr. LAMBERT LACK said he sympathized with Dr. Grant's remark. He had seen one or two cases which had been operated upon by skilled operators, yet the bands were there, and the nasal obstruction persisted. The cases presented all the symptoms of a recurrence of the adenoids. Until a remedy was discovered, he suggested that a little less operative zeal in that corner might prevent a few of them.

Dr. PETERS replied that he thought the bands were partly operative and partly natural. He thought the bilateral symmetry pointed to the natural condition, which was not very infrequent, whereas the cicatricial part pointed to the post-operative effect. He thought the fossa of Rosenmüller had not been cleared up, and so cicatricial tissue had been added to the natural condition.

General Infiltration of Larynx, probably Luetic.

By DAN MCKENZIE, M.D.

THE patient, a woman aged 44, had suffered from hoarseness, with occasional loss of voice, for the last nineteen months, and was subject to distressing paroxysms of coughing and severe dyspnœa. These

attacks were set up by swallowing food, and so her meals have been seriously restricted in quantity. Purulent discharge from right nostril; in July and September of last year two sequestra were washed out of the nose on syringing.

When she came to hospital a month ago the laryngeal infiltration simulated tuberculosis; the epiglottis was thickened, and lobulated masses invested with muco-pus were visible in the interior of the larynx, particularly in the neighbourhood of the right ventricular band. There was pain on swallowing. Von Pirquet reaction negative; no signs in lungs; no tubercle bacilli in sputum. The right nostril contained crusts, and the inferior turbinal has disappeared. The Wassermann reaction was negative (Dr. Wyatt Wingrave), but the therapeutic test seems to favour the diagnosis of syphilitic infiltration, seeing that potassium iodide in 10-gr., and, later, 15-gr. doses has effected considerable improvement in the condition of the larynx, and along with this improvement it has been noticed that the resemblance to tuberculosis has become much less marked.

The PRESIDENT said the result seemed to confirm the diagnosis of tertiary syphilitic infiltration.

Destructive Ulceration of the Hard and Soft Palate in a Young Male Adult, with Exfoliation of Large Mesial Sequestrum, apparently of Specific origin, in a Tuberculous Subject.

By J. DUNDAS GRANT, M.D.

THE patient is a man aged 24, with an oval-shaped perforation of the hard and soft palate, with thickened deep shelving walls which are rough, irregular and bathed with sticky pus. It is somewhat funnel-shaped, and the floor at the deepest part is sloughy. It commenced as a small white spot on the roof of the mouth about six months ago, and has gradually increased in spite of his medical attendant having treated him freely with mercury and iodide of potassium. About four years previously one testis was removed on account of tubercle, and one and a half years later he had apparently tuberculous disease of the elbow. He had an ulcer, presumably tuberculous, on the uvula about twelve months ago, and the uvula was removed. He has been losing flesh for a year, but has no cough; the chest is free from all signs of tuberculosis. On

inquiry there is no definite history of specific inoculation, but there have been opportunities for its occurrence. No tubercle bacilli or *Spirochæta pallidæ* have been found. He has again been ordered perchloride of mercury, iodide of potassium, and good feeding. On December 1 the perforation was found to have extended in depth and a large sequestrum of bone was detected and removed. Nitrate of silver was applied to the edge of the perforation. On January 2 he was admitted into the hospital on account of the occurrence of hæmorrhage from the anterior part of the ulcer on the previous day. Chloride of calcium was ordered, and the plug which had been introduced was removed without any recurrence of bleeding. Calmette's ophthalmic reaction was found negative, but Wassermann's serum test positive. He has again been ordered iodide of potassium in 10-gr. doses, and a mouth wash of acetico-tartrate of alumina. It is proposed to give him intramuscular injections of calomel. Apparently we have to deal with a specific ulceration in one who was formerly the subject of tuberculosis. Evidence has been forthcoming as to the probable inheritance of a specific dyscrasia, of which, however, the patient presents none of the usual physical signs.

Growth in the Nasopharynx of an Elderly Female, like very pale "Adenoids," but much more Dense in Consistence, suggestive of Malignancy.

By J. DUNDAS GRANT, M.D.

THE patient, a woman aged 63, was referred to the exhibitor on the 29th of last month on account of swelling of her right tonsil and obstruction in the nose. No air could pass through the left nostril, although the front part of it was quite free. On posterior rhinoscopy the nasopharynx was seen to be occupied by a growth which to the eye presented the appearance of adenoids, but to the touch was very much tougher, and a portion was removed for examination under the microscope with the expectation that it would present the characters of epithelioma. On a second attendance she was ordered iodide of potassium, on the presumption that the condition might be tertiary specific. She was so intolerant of examination that palpation was not then practised, but a probe passed from the anterior naris impinged upon bare or very thinly

covered bone. The disease was apparently of four months' duration. During this time her vision has been getting defective, and the left eyeball has turned inwards—paralysis of external rectus. An ophthalmological examination will be made.

Microscopical examination revealed the structure of a granuloma, and there is every probability that the case is specific and that a deep-seated sequestrum will be detected.

DISCUSSION.

Mr. HERBERT TILLEY asked whether Dr. Grant had examined the patient with his finger. He had had to do with a stout lady, aged 53, from whom he removed the largest adenoid he had ever seen.

Dr. SCANES SPICER said he caught sight of some thick, whitish-grey pus, very much like little masses of diphtheritic membrane. He asked whether sinuses, as foci of infection, had been definitely excluded.

Dr. GRANT, in reply, said the patient was very intolerant of examination. The tissue was denser than adenoids generally were, and he removed a small portion with StClair Thomson forceps. Dr. Wingrave reported it as lymphatic infiltration, with plasma cells, stratified epithelium and glands. There were no nests and no sign of malignancy. He would have her eye examined, and in the meantime she was having iodide of potassium. He believed the finger would come upon bare bone.

Microscopical Section from Fragment of an Adenoid-like Growth in the Nasopharynx of a Middle-aged Lady, probably an Early Stage of Epithelioma.

By J. DUNDAS GRANT, M.D.

THE patient's chief complaint was of dullness of hearing in the left ear, there being great difficulty in the introduction of a Eustachian catheter. A year ago what appeared to be 'adenoids' were removed, and it was found that the nasopharyngeal space was encroached upon by a projection of the atlas. A return of the stuffiness in the nose led to a renewed examination. The adenoid growth seemed to have redeveloped, and a portion of the tissue was removed, giving relief to the breathing through the nose. There are no enlarged glands, but the

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microscopical examination of the fragments recently removed (shown on the slide at present under the microscope) are considered by Dr. Wyatt Wingrave to present signs of early malignant disease.

Laryngeal Growth, probably Malignant, in a Man aged 61.

H. W. FITZGERALD POWELL, M.D.

THE patient, a male, aged 61, came into hospital on December 23, 1909. He complained of loss of voice and some difficulty of breathing. On examination he was seen to be suffering from some inspiratory stridor, and the voice was nearly lost.

The larynx: The left ventricular band is occupied by a broad-based swelling, which was commencing to ulcerate in centre, where there is a small greyish patch. The left vocal cord is entirely fixed, and has no movement on phonation; the cord is ragged on this side and thickened. The left arytaenoid and aryepiglottic fold are swollen and somewhat oedematous; the growth appears to extend into the pyriform fossa: there are no glands to be found enlarged in the neck. The whole of the left side of the larynx appears to be involved in the growth.

History of case: Patient states that a few years ago he noticed some trouble in singing. For twelve months his voice has been husky, which has got much worse the last few months; has been troubled with shortness of breath, much worse on exertion, for some weeks, and thought he would suffocate at times. There is no history of, or visible manifestations of, specific disease.

Since admission: His general condition and breathing have improved, but appearance of growth is unaltered. He has been put on potass. iod., 20 gr., three times daily for two weeks.

The PRESIDENT said that, although it had only slowly developed, there was no doubt about its malignancy. Possibly the patient would live longer without an operation than if one were performed.

Laryngological Section.

February 4, 1910.

Dr. J. DUNDAS GRANT, President of the Section, in the Chair.

Two Cases of Foreign Bodies : (A) in Lung, (B) in Œsophagus.

By H. J. DAVIS, M.B.

(A) *A piece of bone in right lung. Pulmonary abscess; empyema : operation; foreign body coughed up six weeks later—i.e., six months after its entry into the bronchus.*

A MAN, aged 53, was admitted into the hospital under the care of my colleague, Dr. Seymour Taylor, with physical signs pointing to empyema and pulmonary abscess of the right lung. Five months before this, while eating ox-tail soup, "a piece of bone slipped into his windpipe"; he coughed violently, choked, and thought he was dying, but as he did not do so, he continued his meal. Beyond occasional paroxysms of coughing, nothing happened for five months, when he developed pleurisy; a week later he was admitted into the hospital with the condition above described. A rib was resected. The empyema communicated with a large abscess cavity in the lung, which was drained; the discharge was so profuse that the wound was dressed three times a day. The patient was so ill that he could hardly be moved, but on being turned over on his side, preparatory to a dressing, he coughed violently, "felt the bone in his throat and spat it out." His condition rapidly improved and he left the hospital well. The bone and a photograph were exhibited.

- (B) *A pin, $1\frac{1}{2}$ in. long, impacted in the oesophagus; pin grasped with forceps but could not be moved; oesophagoscopy negative: pin passed into stomach and vomited up the same night on recovery from anæsthetic.*

THE patient was a soldier, aged 30. "He went to sleep on a sofa with a large pin between his teeth; it suddenly slipped down, and he could feel it sticking into his throat at the root of the neck." Attempts had been made at two hospitals to remove it before he came to the West London. Mr. Morton took a skiagram, and the pin could be seen head downwards with the point resting against the sixth vertebra. Under chloroform the pin was grasped with forceps, but, in spite of all manipulation, it could not be removed, and, fearing to break it, he was sent back to the ward with $\frac{1}{4}$ gr. of morphia injection. Later, oesophagoscopy detected the exact point of impaction, but the pin could not be seen. A second skiagram showed everything clearly except the pin, which it was assumed must have been swallowed. On recovering from the anæsthetic he was very sick: "he felt the pin fly up into his throat, and he took it out with his fingers."

The foreign body and a skiagram were exhibited.

DISCUSSION.

Dr. W. HILL said he had found that merely passing an instrument often released the foreign body; in fact the operator was often baulked of his quarry after passing the oesophagoscope, for directly the cricoid was pulled forward by the instrument the body was released if impacted high up. He was called one afternoon to hospital to deal with three people—a man, a woman, and a child—each of whom had a foreign body in the cervical oesophagus. In the two adults the body was a mutton bone, and while passing the tube through the introitus the patient in each signalized that the bone had moved down. The foreign body in the third case, a child of six, was a safety-pin, open, with the point above. A Killian tube (9 mm. diameter and 40 cm. long) was passed, under chloroform, through the oesophagus and into the cardiac end of the stomach, but no pin could be seen. A further X-ray examination was then made, and the pin was seen in the pyloric antrum. As the pin was large and open, he left it to a surgical colleague to decide whether or not it would be safer to perform gastrotomy or to trust to the pin being passed *per rectum*. Unfortunately, gastrotomy was not performed until three hours after the last X-ray examination, and the stomach was then found to be empty. Later it was safely

defecated. Most foreign bodies which had managed to pass through the phrenic constriction of the gullet might be relied on to pass safely through the entire length of the gut in time, and in view of what happened in this case he would in future adopt an expectant attitude, even in the case of a large open safety-pin in a small child, if it passed into the gullet. The case of an ordinary pin or a needle in the stomach was quite another matter, however, and Dr. Davis was lucky in that he had been relieved of the difficulty of facing this problem by an opportune attack of vomiting so successfully coming to his aid.

Mr. TILLEY desired to mention the ease with which a bronchoscope could be passed over a foreign body so that it might be missed altogether. In the collapsed state of the œsophagus it was easy for a pin to lie in a fold. That occurred in a case of his in which a safety-pin fell from a mother's blouse into a child's mouth, was swallowed, and lodged in the gullet with the point upwards. In drawing out the œsophagoscope he caught hold of the curved portion of the open safety-pin, and managed to swing it round in the œsophagus and to extract it.

Dr. DAVIS replied that he seized the pin with straight forceps, but he could not have extracted it without breaking it. The pin was eventually vomited up. After passing such instruments there was always some abrasion of the surfaces, and he gave formalin 1 in 1,000, with glycerine, in doses of one teaspoonful every hour. That, swallowed slowly, assisted healing, and relieved the immediate inconvenience following the operation. The pin, as could be seen, was considerably bent.

Fracture of Hyoid Bone in a Man aged 56 ("Garrotter's Throat").

By H. J. DAVIS, M.B.

THE patient came to the hospital complaining of a sore throat, "as the front wheel of a van had passed across his neck a week ago." Laryngeal and faucial examination showed nothing; no swelling and no lesion. On grasping the neck or swallowing he complained of great pain. The skiagrams, exhibited, showed quite plainly a fracture of the body of the hyoid bone.

Pemphigus Vegetans affecting the Larynx and Fauces, Nasal Cavities, Tongue, Cheek, and Lips of a Woman aged 40.

By H. J. DAVIS, M.B.

THE case was shown at the last November meeting.¹ The patient left the hospital very much improved, but she had lately had a fresh outbreak on the skin. This was accompanied by bronchitis (? spreading down of the bullæ into the tubes) and an outbreak in all other areas of the respiratory tract.

DISCUSSION.

Dr. STCLAIR THOMSON said some members might be disappointed that there were not more pemphigus blebs to be seen, but he had seen the case when these were much more marked. In a previous discussion on the matter it was pointed out how many of the cases ended fatally.

Dr. W. HILL asked what the general health of the patient was. In these cases the patients usually became very ill and soon died. Also what treatment was adopted?

The PRESIDENT (Dr. Dundas Grant) said a surgeon of great repute was once criticized in the press for having kept a patient having the disease for a couple of weeks without treatment by arsenic in order to show the effect to students, so sure was he of its efficacy.

Dr. DAVIS replied that when the patient was in the hospital three months ago, she was admitted under the care of Dr. Abraham, who treated her with large doses of arsenic. She got well temporarily. But three weeks ago she had another acute attack, with blebs all over the body. She also had bronchitis, and was very ill. The only local treatment he had given was pure glycerine of boracic acid as a mouth-wash and to swallow. First of all he had tried orthoform, but the patient stated that she had derived much more relief from the glycerine. He believed she would recover from this attack as before, but would have another. The disease invariably ended fatally.

¹ *Proceedings*, iii, No. 2, p. 22.

A Case of Laryngeal Vertigo in a Man aged 47.

By H. J. DAVIS, M.B.

THIS patient was transferred to me by my colleague, Mr. Dunn, from the eye department. His condition when first seen a week ago was as follows: (1) Severe subconjunctival hæmorrhages; (2) hæmorrhage from dilated veins in pharynx; (3) hæmorrhage into both vocal cords; (4) right inguinal hernia; (5) numerous bruises, scalp wounds, &c., the result of falls. He states "that a tickling begins in the throat, he coughs and coughs for five to ten minutes, he then becomes violently giddy, and he falls to the ground insensible; when he recovers in a minute or two all desire to cough has gone and he feels all right." He had his "uvula removed on Christmas Day after a severe attack" (this has been very liberally done, as the stump is flush with the palate), and he has been a little better since. In dry weather he is worse—he is a pipe smoker—"but the mere smell of cigarette smoke in a room or in the street brings on an attack at once." Though he may have several severe coughing paroxysms in the day, "he has never fallen down unconscious more than once during the day."

The patient is a big, heavy-looking man, with a very red face. The lungs are quite normal; and, beyond an accentuated aortic second sound and exaggerated knee-jerks, there are no signs of disease; no history of epilepsy in the family. The larynx, beyond the hæmorrhages on the cords, is normal; the pharynx is red and congested. The condition is a very distressing one, and any hints with regard to treatment will be welcome, as he cannot follow his occupation.

I ordered a mixture of syrup codeinæ ʒi, acid hydrobrom. dil. ʒi, and aq. chlorof. ad ʒi, and this has given him a little relief—not much.

DISCUSSION.

Dr. WATSON WILLIAMS said it was an interesting example of a somewhat rare condition. One point to which he desired to draw attention was the curious fact that these cases did not seem to have inspiratory stridor; it was entirely expiratory spasm, and therefore not a simple laryngeal spasm. This patient thought it was caused by a tickling cough; then he got the sudden vertigo, fell down, and sometimes was unconscious. He (Dr. Watson Williams)

did not think anyone had given a convincing explanation of the pathology of the cases of laryngeal vertigo. One point he had ascertained in regard to the patient just seen which was not mentioned was that six months before the first attack he had a severe accident. He was riding on a cart when he fell off and was dragged some distance, causing a severe blow on the head, which indented his skull. He was unconscious for some time. Possibly the shock had something to do with such laryngeal conditions. Dr. Watson Williams had a patient, a boy at school, who had an attack if there was sudden pressure over the laryngo-tracheal region. Dr. Davis's case, like many others, seemed to have increased micturition following the attacks. He suggested that the only thing to do was to continue building up the general health and treat it as a neurosis, giving bromide and arsenic, with the idea of controlling the onset of the attacks. It was good that smoking should be discontinued, and the patient should have as much fresh air as possible.

Dr. DE HAVILLAND HALL suggested that, as the attacks were generally produced by a dry, irritating cough, the patient should be given 5 gr. of iodide of potassium with 8 gr. of chloride of ammonium and extract of liquorice three times a day. That was particularly good where the tension was high, as it was in the present case. He would also order a linctus containing a small quantity of liquor trinitrini (℥ $\frac{1}{3}$) with camphor.

Dr. PEGLER said that a simple but not very well known remedy for irritation in the throat consisted of a combination of 1 dr. of tincture of catechu in 1 oz. of compound tincture of benzoin; 15 drops to be taken on a lump of sugar three times a day. He asked what was the character of the uvula before amputation, and if there were any reason to attribute the attacks to that organ. Several years ago he had a very severe case of the same nature, in the person of a railway guard. It was very serious, as he used sometimes to fall down unconscious when on duty, and was in great danger of falling on to the line. He reduced the elongated uvula to the normal dimensions, and the man had entirely recovered from his vertigo. He saw him again two or three years afterwards, and learnt that the good result was permanent.

Dr. FITZGERALD POWELL thought that this was a very interesting case, the causation of which, since it was first described by Charcot, was very uncertain. Dr. McBride thought the vertigo was caused by the forced expiration against the cords closed by spasm. He (Dr. FitzGerald Powell) suggested that there might be some focus in the nose, causing reflex irritation, because it was said that when the patient inhaled cigarette smoke it set up cough and spasm. Sir Felix Semon had reported a case of spasm of the larynx which he treated successfully by means of a daily spray of a 2-per-cent. solution of cocaine. The tickling cough was said to be relieved by citric acid, glycerine, and syrup of red poppies.

Dr. SCHOLEFIELD said he remembered a somewhat similar case in a gentleman aged 85. At the time there were children in the house suffering from

whooping-cough, and he regarded the case as one of whooping-cough in a very old subject, and thought the feebleness of the heart had something to do with the attacks. As the case progressed it seemed that only three or four little coughs were necessary to cause the attacks. On many of the occasions epileptiform attacks took place, so that death seemed to be imminent. But in the course of six months the condition wore itself out; this seemed to coincide with the whooping-cough view. The present patient had exaggerated knee-jerks, and ankle clonus was present.

The PRESIDENT said that in some such cases there was syncope rather than vertigo. He thought all would agree that the condition in this patient was some disturbance in the cerebral circulation due to the cough; there was heightened tension, as Dr. de Havilland Hall had already remarked. So that treatment directed to lowering this, rather than invigorating treatment, was indicated. Would Dr. Davis say whether the urine had been examined? Possibly the patient was a candidate for cirrhotic disease of the kidney.

Dr. WATSON WILLIAMS said he did not think that it was correct to say that Sir Felix Semon had success from cocaine applications with a similar case of laryngeal vertigo, and considered that the reference to any such supposed case should be given.

Dr. DAVIS replied that the case was a good example of laryngeal vertigo, and the manner in which the attacks came on was typical. Not every form of irritation caused an attack. He had seen him in one attack in the out-patient room; he was unconscious, but he did not micturate, nor was he sick nor did he bite his tongue. His uvula was said to have been very long, but it had been removed before he saw the patient. The patient was a butcher, and he complained that the appearance of his eyes and face deterred customers from entering his shop, and his business suffered in consequence. There was no albumin in the urine.

Large Tuberculous Ulcer on the Right Side of the Tongue in a Man aged 42.

By H. J. DAVIS, M.B.

THE patient has laryngeal tuberculosis and also physical signs of chronic phthisis at both apices; he was sent to me by my colleague, Dr. Pritchard. There is a large, flat, oval ulcer on the right side of the tongue which might easily escape detection. The position is an unusual one, for in tuberculosis the dorsum and tip of the tongue are, as a rule, the favoured areas. The patient does not look ill, and he is not

wasting, but he can only eat with great difficulty. The pain in the ear of which he complains is at once relieved by applying cocaine to the ulcer.

DISCUSSION.

Dr. WATSON WILLIAMS said he would like to know on what evidence it was said to be tuberculous. It might be a septic ulceration.

The PRESIDENT said there was much induration at the base of the ulcer, and had it not been for the statement as to its tuberculous nature, he would have inclined to another diagnosis. The position was an unusual one for such an ulcer; they almost invariably occurred on the dorsum. Were there any changes in its character during the time of observation?

Dr. DAVIS replied that he regarded the condition as now somewhat improved. When he first saw the patient he thought it might be malignant disease, but there was no limitation of movement of the tongue, and there were no enlarged glands. Moreover, there was pain on even touching the ulcer with the finger, and there was also pain in the ear. He did not think that either gumma or malignant disease ever caused so much superficial pain as that. He had had no scrapings taken, but it looked like a tuberculous ulceration, very shallow and very flat. He had treated it with orthoform and chromic acid, 40 per cent.; this formed an albuminate over the ulcer and protected it.

Leucoplakia (?) of the Soft Palate.

By ANDREW WYLIE, M.D.

THE patient, a man aged 67, complains of pain on taking solid food for the last four months. He is otherwise well. There is no loss of weight, no glandular enlargement, and no specific history. The patient smokes $\frac{1}{2}$ oz. of tobacco per day, and, having no teeth, inserts the stem of his pipe a considerable distance into his mouth. On examination, a superficial ulcerated spot about the size of a sixpence is seen on the right side of the soft palate, extending to the middle line. On the edge of the ulcer, especially on the right side, is a whitish, horny epithelium, which was more apparent three weeks ago. The palate moves freely, and on palpation there is no hardness. The case has been under observation for three months, and although treated by local application of chloride of zinc, internal administration of potassium iodide, a soft diet, and total abstinence from smoking, the condition does not improve.

The exhibitor considered that this case resembled a leucoplakic condition of the mucous membrane, described by Butlin and others as terminating in epithelioma, and had delayed removing a piece for microscopic examination until the case was shown to the Section.

Dr. Wingrave had examined a surface scraping of the ulcer, and reported that there were no tubercle bacilli or spirochætes.

DISCUSSION.

The PRESIDENT said the case was on all fours with one which he brought before the Section recently, which he had been, and still was, at a loss to name; he was properly criticized for calling it erythema. There was an excoriation, and around it were patches of leucoplakia over the front of the palate. His patient was a great smoker, and the same was true in Dr. Wylie's case. This patient, on account of the absence of teeth, had to put the tip of his pipe a long way in his mouth, and it impinged on that portion of the palate. He did not think there was any malignancy in the present case.

Dr. DONELAN said that while etymologically it would be correct to describe this white patch as leucoplakia, it was not, he thought, the condition described by Mr. Butlin as so prone to undergo an epitheliomatous degeneration. For this, which occurred predominantly on the tongue and in females, Mr. Butlin had suggested the term "leucoma." Dr. Donelan thought the present case one of aggravated smoker's patch, which appeared to be about to become an epithelioma, though, of course, in the absence of a microscopic report, any definite opinion would be premature.

Dr. WYLIE replied that he intended to remove a piece in order that a microscopic section might be made.

Rare Congenital Deformity of the Nose in an Infant.

By GEORGE WILKINSON, F.R.C.S.

THE infant was 8 months old at the time the photograph was taken. The deformity consists of a deep depression in the middle line of the nose, with wide separation of the nostrils, and flattening and broadening of the whole feature. The nose is 3 cm. wide at the level of the alæ, but only projects about 1 cm., the greatest projection being on either side of the middle line in front of each nostril. These two prominences are separated by a depression of the tip of the nose 2 cm.

wide. The nasal bones and nasal processes of the superior maxillæ are flattened. There is no separation between the nasal bones. The columella is 2 cm. broad, and the anterior nasal spine can be felt behind the columella as a broad projection of bone, about $1\frac{1}{2}$ cm. from side to side.

On inspection of the nasal passages the anterior ends of the nasal septum can be seen as a prominent ridge on the inner side of each vestibule. The two sides of the septum are evidently separated from each other. There is no nasal obstruction. On everting the upper lip there is seen a distinct notch on the buccal surface in the very centre of the lip. There is also a well-marked notch in the middle line of the alveolar process. The two halves of the alveolus are not in alignment,



Congenital deformity of the nose.

but meet with a forward pointing angle. Two uncut incisors can be felt under the gum on either side of the mesial notch, showing that this represents a division between the two halves of the premaxillary bone.

With regard to the developmental cause of the deformity, it has arisen, no doubt, from failure of fusion of the two mesial masses of the fronto-nasal process. The mesial masses usually coalesce at an early period of fetal life to form the anterior part of the nasal septum, the columella, the median segment of the upper lip (lunula), and the premaxilla. Failure of union of these mesial masses seems to be a rare condition in the human subject.¹ Cases of median cleft of the lip (a true

¹ Professor Keith, "Congenital Malformations of the Face," *Brit. Med. Journ.*, August 7 and 14, 1909.

hare-lip) due to this cause have been reported by Mr. Clutton and Mr. Edmund Owen. Mr. Bland-Sutton instances the median split in the columella of Parisian pugs as a similar instance of want of union (Professor Keith). Professor Keith states that there is not a single museum specimen in London to illustrate the occurrence of a fissure in the mid-line of the nose in the human subject, nor is there a specimen of dermoids or of fistulæ which occasionally occur in the line of this fissure.

DISCUSSION.

Dr. WATSON WILLIAMS said that a similar case in an adult was reported by Mr. Stewart some years ago.

Dr. W. HILL asked what Mr. Wilkinson intended to do for the case. Mr. Stewart operated upon his patient by making a vertical incision through the groove, removing redundant tissue, and bringing the two halves of the tip of the nose together. The result was a strikingly good one.

Dr. DAN MCKENZIE asked what was the condition of the septum. In view of the recent discussions with regard to the effect of removal of the septum in submucous resection, the condition of the septum in this case might be of interest.

Dr. PEGLER produced a volume of the *Proceedings of the Laryngological Society of London*¹ containing a figure and description of the late Mr. W. R. H. Stewart's case of the same kind in a young man.

The PRESIDENT asked what was the most appropriate time for performing such an operation: now, or when the nose had developed further?

Mr. WILKINSON replied that the difficulty in operating on the cases was just what had been spoken of. The septum was much broadened and the septal cartilages could be felt inside the nares. His first idea was to make an incision in the mid-line of the nose, dissect up the skin and suture the lateral cartilages in the middle line, but he found that would block the anterior nares and reduce them to oblique slits. At present he had had a truss made for the child, after the fashion of Hainsby's truss, to make pressure on the malar bones and nasal processes of the superior maxillæ. By wearing this constantly the nose might be narrowed, and, later on, an operation might be done of sewing the cartilages in the middle line.

¹ 1896-7, iv, p. 43.

**Specimen of Lymphosarcoma of the Tonsil, removed by
Lateral Pharyngotomy after Vohsen's Method.**

By GEORGE WILKINSON, F.R.C.S.

FROM a married woman, aged 37. Two months' history. The tumour was the size of a large walnut occupying the right tonsil, and filling the right half of the fauces. The mass was well defined from the surrounding tissues, but firmly fixed, and was decidedly tender to touch. No palpable enlargement of the glands. After preliminary clearing of the mouth of septic teeth, the patient was operated upon on October 13. Semi-reclining position. Chloroform. Incision from tip of mastoid to great cornu of hyoid, then curving forwards and upwards for one inch. External carotid isolated, and the lingual, facial, and ascending pharyngeal branches ligatured. The jaw was divided in front of the attachment of the masseter, and the ascending ramus pulled strongly forward over the horizontal ramus (Vohsen's procedure). There was now good room to work towards the pharyngeal wall, and the tonsil was readily dissected out and removed, along with two slightly enlarged glands which overlay it. The pharyngeal wound was sutured with catgut, and the jaw with silver wire. The pharynx healed in a fortnight without suppuration or any escape of fluid from the mouth through the wound. After a fortnight an abscess formed over the jaw, and the silver wire and a small sequestrum had subsequently to be removed. The microscope showed the tumour to be a lymphosarcoma.

**Lupus of the Nose, Hard Palate, Fauces, and Epiglottis, in
a Girl aged 10.**

By W. STUART-LOW, F.R.C.S.

THERE were two interesting features in this case: First, the early implication of the epiglottis; secondly, that the child's father was living and healthy, but that the mother's first husband succumbed to phthisis pulmonalis.

DISCUSSION.

Dr. VINRACE asked what Mr. Stuart-Low meant by early implication of the epiglottis—early in disease or early in life, or both? There was a patch of lupus on the thigh, which confirmed the nature of the condition, if any confirmation were necessary. He also asked as to the order in which the symptoms appeared, and the dates.

Dr. KELSON said that in June, 1904, he showed a case of similar character—a boy aged 12, whose epiglottis he cured by the galvano-cautery. But the condition appeared on the gum a short time after the epiglottis got well. The effect of arsenic on these cases was remarkably good. He sent his case to the London Hospital for the light treatment to be applied to the gum.

Mr. BARWELL suggested that before the application of the galvano-cautery, arsenic and general constitutional treatment, preferably at a sanatorium, should be carried out. After such treatment many of the cases required no local measures at all; and if local treatment were eventually necessary, the disease would be by then much more limited in extent than it was at present.

Mr. STUART-LOW, in reply, said he referred both to the early disease and the youth of the patient. It began at eight years of age, when the child used to crawl about the floor in the room where the mother's first husband had died of phthisis. Probably the child scratched her nose, and the bacilli were thus implanted. He omitted to mention that there was a large patch of lupus on the thigh and one on the back. He intended to give arsenic and afterwards to follow Dr. Kelson's advice.

**Extensive Tuberculosis of the Larynx in a Middle-aged Man,
with Rapid Evolution, showing almost Complete Recovery.
Galvano-caustic Treatment.**

By J. DUNDAS GRANT, M.D.

THE patient, a man aged 55, was first seen by the exhibitor in July, 1909, complaining of something forming in the left side of his throat, thickness of voice, and a pain during swallowing running up to the left ear, so great as to make eating and drinking, as he said, impossible. His illness began with an attack of influenza in December, 1908, and with hæmoptysis (to the extent of half a gallon) in March, 1909, from which time the throat became dry and gradually painful, while the voice became hoarse. The exhibitor's colleague, Dr. Fenton, found

dullness of both apices and crepitations at the right apex, and the patient had diminished in weight from 10 st. 4 lb. to 8 st. 7½ lb. There was no family tendency to tuberculosis, but on inquiry it was elicited that, in his business as a house-painter and paperhanger, he had had to work in close contact with a man who was apparently the subject of pulmonary tuberculosis.

When he was first seen there was enormous infiltration of the epiglottis, of both aryepiglottic folds and particularly of the left ventricular band, on which there was a well-marked irregular ulcer, while at the junction of the epiglottis with the right aryepiglottic fold there was a circumscribed mass of infiltration amounting to a tuberculoma. The condition looked an extremely hopeless one, but the exhibitor decided to try the effect of the galvano-cautery, and this was applied freely into the ulcer on the ventricular band, while two galvano-caustic punctures were made into the epiglottis. The patient received almost instant temporary relief from his pain, and he was ordered to inhale into his larynx, by means of Leduc's tube, a powder consisting of equal parts of anæsthesin and orthoform. This relieved him at first only for a few minutes at a time, but sufficiently to enable him to consume a small meal. Later the relief lasted for an hour, then from one meal to another, and of late he has been able to eat and drink without it, though he finds it advisable to use it once a day. From the time of the first cauterization, on July 20, to August 10 the pain was considerably diminished. There was then still considerable, though less, infiltration of the epiglottis; the tuberculous swelling had diminished, but close to its site there was a distinct loss of substance, in fact an irregular ulcer. The galvano-cautery was then applied to this ulcer, and punctures were made into the epiglottis and ventricular band. In the beginning of September the cauterization was repeated, and after this the pain gradually died away.

At the present time the epiglottis has returned nearly to the normal thickness, but is somewhat distorted by the cicatrix on its left side at the spot where the tuberculous ulcer previously existed. There is now no ulceration whatever; both cords are visible; there is a slight infiltration of the aryepiglottic folds, but they are apparently quite soft and elastic. The patient has been at work all the time, and there is every reason for supposing that his recovery is to a great extent attributable to the local treatment. The case is brought forward as offering encouragement, even when the laryngeal condition is of considerable extent; probably the absence of a family constitutional tendency and the "accidental"

nature of the infection were favourable elements in the case. The patient was the father of thirteen children, of whom six died in early infancy, the survivors, however, being in sound health. There is no history of specific infection.

DISCUSSION.

Dr. FITZGERALD POWELL said he had found much reaction after applying the galvano-cautery in such cases if applied often. He asked how often Dr. Grant applied it; also whether he thought there was any danger in applying it to the arytenoid in cases of disease. He also asked how long Dr. Grant continued the treatment.

The PRESIDENT replied that he allowed two or three weeks to elapse between the applications. He used puncture; the point could be introduced and then rotated a little, so as to cause a funnel-shaped burn. He had been surprised at the absence of reaction, and it seemed strange that it should produce relief almost immediately. It was difficult to say how long the treatment should be continued; he did not propose to make any further applications in the present patient. One sometimes avoided using it, or stopped using it, because of the asthenia from the general disease.

Extensive Fracture of Nasal Septum, complicating Maxillary Suppuration, in a Woman aged 39 ; Operations ; Recovery.

By JAMES DONELAN, M.B.

THE fracture was caused by a fall about twelve years ago. The patient was seen first last summer on account of suppuration in the left maxillary antrum. The septum was much deflected in a most irregular manner and in separate fragments, chiefly to the left. One of these fragments can still be seen in the right side, forming until recently a large synechia with the right inferior turbinal. The left side was corrected by a series of operations under local anæsthesia to the extent now seen, and the antrum successfully drained by removal of portion of nasal wall. Several synechiæ were dealt with in the course of this treatment. The large one on the right side was incised last week, and, the separation having been maintained, it is now proposed to freshen the adjacent surfaces and fix it to the rest of the septum. Incidentally this patient, who was disfigured by a chronically congested and swollen nose, has benefited much in point of appearance.

Extension of Cuticle inside the Nostrils of a Man aged 41.

By JAMES DONELAN, M.B.

THE condition caused much discomfort when indoors from dryness and crusts. The patient is not affected in the open air and finds temporary relief from the use of vaseline. The condition has lasted over twenty years. He suffered a good deal from chronic nasal catarrh in childhood and youth, and has general hypertrophic rhinitis and a deflected septum. History of syphilis fourteen years ago.

DISCUSSION.

Dr. PATERSON said the man had some erythema of his face, and probably it was seborrhœic. The condition seemed to have extended into the skin of the vestibule. There were fissures and cracks in it. On the other hand, the mucous membrane of the nostril was dry and injected—a condition not infrequently associated with dryness of the vestibule.

Dr. PEGLER asked on what ground there was thought to be an extension of the cuticle into the vestibules. He only noticed some dry rhinitis in Kiesselbach's area on the left side.

A Case of Paresis of Palate, Pharynx, and Œsophagus.

By W. H. KELSON, M.D.

A. C., MALE, aged 26, a clerk, was attacked with sore throat fourteen days before Christmas. About Christmas-time food and drink began to come back through the nose; liquids particularly at once set up coughing and returned; his voice also was reduced to a whisper. Notes on admission to hospital, January 21: Pulse irregular, 84; temperature 98.4° F.; lungs normal. Palate moves, but very slightly; pharynx anæsthetic and motionless. Vocal cords move well, but look red and swollen; glottis elliptical. A quantity of muco-pus hangs about the upper opening of the larynx. Feels generally weak, but knee-jerks present. No eye symptoms; urine contains a small quantity of albumin; swabs show no Klebs-Loeffler bacilli. January 18: Patient is taking liq. strychninæ miiij, and is rather better.

DISCUSSION.

The PRESIDENT said it seemed to be a post-diphtheritic case, with singular absence of ocular symptoms.

Dr. H. J. DAVIS recommended that the strychnine should be injected subcutaneously in these cases. An injection of *Mij strychninæ nitratis* once a day would suffice. The nitrate was a more efficient salt than the hydrochlorate. Strychnine injections were painful, but more benefit would be derived than by administering strychnine in a mixture. The condition was post-diphtheritic.

Dr. WATSON WILLIAMS said he had recently seen a similar case following a sore throat; and he thought such cases were always due to the diphtheria bacillus. His patient recovered under the influence of arsenic and *nux vomica*.

Dr. KELSON replied that the evidence of it being diphtheritic was very slight; indeed, it seemed to fit the supposition that it was post-influenzal.

**Congenital Occlusion of the Left Posterior Nares in a Girl
aged 19.**

By DAN MCKENZIE, M.D.

THE patient had never been able to breathe through the left side of the nose. On anterior rhinoscopy the septum was found to be deviated to the left, and on posterior rhinoscopy the left choana was seen to be completely obstructed. Acting upon the advice of Dr. Dundas Grant, the exhibitor first of all performed submucous resection, removing the vomerine segment of the septum as far back as he could reach. Some weeks later the choanal diaphragm, which proved to be osseo-membranous in structure, was cut through with a chisel, and the opening so made was enlarged with a burr, under the guidance of the finger in the nasopharynx, to the approximately normal dimensions of the choana. A large-sized drainage-tube inserted through the breach the day after the operation proved to be intolerable and had to be removed after twenty-four hours. Nevertheless, the result has fully realized expectation, and the patient can now breathe comfortably through both sides of the nose.

DISCUSSION.

Dr. PATERSON asked whether a cast might be made of the upper jaw, as it would be interesting to compare the two sides with regard to their development; perhaps on a future occasion it could be laid before the Society. In a

similar case of his own he was having a cast made, as there was considerable interest in determining the changes which had occurred. It was also a unilateral case. In the present case the differences were not very obvious, but he would not like to express an opinion without seeing a cast. In nearly all unilateral cases the hard palate stood somewhat higher on the affected side.

Mr. BARWELL said he showed a similar case in a girl eighteen months ago, in whom there was no obvious asymmetry of the jaw, but no casts were made. In his case he did a submucous resection, and removed a large part of the bony septum posteriorly. If this were done, it was unnecessary to use a splint or other dilator. In his own case there was no deafness or affection of the ear on that side, and that was generally found to be so in cases of complete congenital occlusion.

Dr. H. J. DAVIS said that in 1908, at the March and June meetings,¹ he had shown a woman with an almost complete obstruction of the left choana, due to the presence of a thick membrane between the septum and the outer nasal wall. It was considered to be congenital, and he was advised to do a submucous resection in addition to removing the tissue. He did not do this, but freeing the nasal passage amply sufficed to cure the patient.

Dr. MCKENZIE replied that there was considerable deflection of the septum, and it was straightened to render the access to the posterior diaphragm more easy.

Case for Diagnosis.

By ATWOOD THORNE, M.B.

WOMAN, aged 50; looks older. Difficulty in swallowing eighteen years. Nine years ago was told she had stricture of the gullet; tubes were passed and she improved. At intervals the difficulty in swallowing becomes worse, then a "lump comes away from the throat" and she swallows better. On examination, a mass is seen in the posterior pharyngeal wall of the appearance, colour, and size of half a peeled walnut. Case and microscopical specimen exhibited.

DISCUSSION.

The PRESIDENT said it was a most singular case, and he had never seen anything like it. It seemed to be a huge papilloma. He presumed it was not malignant. It might be referred to a committee.

¹ 1908, i, No. 6 (Laryng. Sect.), p. 75.

Mr. ATWOOD THORNE replied that many members thought it was malignant, but the history was against that. It felt like a large wart, firmly fixed; the base was only a little narrower than the head, and it could not be removed with a snare. He intended to remove it, and would then report further.

Hypertrophic Laryngitis with Stenosis in a Syphilitic Subject.

By WILLIAM HILL, M.D.

FEMALE, aged 37. There were evidences of syphilis in the nose, mouth, &c., and there was marked tumefaction of the laryngeal mucosa, causing, *inter alia*, approximation of the ventricular bands and vocal cords; there was also great swelling of the mucosa covering the ary-tænoids and of that lining the deep or postcricoidal pharynx, the latter causing some dysphagia. Owing to the narrowing of the glottis and the accumulation of dry crusts in the interarytænoid region, the patient had suffered at times from attacks of spasm, which were becoming more frequent and alarming. The condition had not yielded to three weeks' treatment by mercury and iodide of potassium, with benzol inhalations, and the exhibitor was seriously considering whether it would not be safer to resort either to intubation or to tracheotomy. He asked for advice, as he was rather uneasy on account of the attacks of spasm with stridor and dyspnœa, but had hesitated to resort to surgical measures in a syphilitic subject.

DISCUSSION.

The PRESIDENT said he thought attention should be directed to the nose. He had seen such conditions due to the inhalation of purulent matter from the nose, and the spasms were generally due to a collection of muco-pus which became inspissated in the larynx and dried during sleep. It was worse in the winter than in the summer. It was benefited by washing out the nose.

Mr. WILKINSON said he asked the patient whether she had crusts from the nose or throat, and she said that during her paroxysms of coughing she forcibly expelled crusts from the throat.

Dr. HILL replied that he had perhaps been remiss in not paying more attention to the local treatment of the nasal condition and in relying solely on internal treatment. He would try nasal syringing, but he very much feared that intubation or tracheotomy would become necessary.

Benign Laryngeal Growth.

By WILLIAM HILL, M.D.

FEMALE, aged 65, with a growth about the size of half a Barcelona nut, which appeared to spring from the anterior two-thirds of the right vocal cord and adjacent floor of the ventricle; phonation was impossible as the growth prevented approximation of the cords. Although the typical appearance of a papilloma was not evident by mirror laryngoscopy, a distinctly papilloid surface could be made out by the nearer view obtainable by direct laryngoscopy; there was no impaired movement of the affected cord. According to the patient's statement, "a papilloma of the vocal cord had been removed by Dr. Felix Semon twenty years ago." An attempt would be made to thoroughly eradicate the growth with the snare and forceps through the exhibitor's large-pattern direct laryngoscope.

DISCUSSION.

Dr. PEGLER said he thought he detected a minutely cauliflower-like surface of the growth, indicating papilloma.

The PRESIDENT said he saw a hint in Garel's recent edition that very often the papillomatous nature of a growth could be settled, when it was doubtful, by throwing it, after removal, straight into alcohol, when the fimbriae showed themselves more distinctly.

Laryngological Section.

March 4, 1910.

Dr. J. DUNDAS GRANT, President of the Section, in the Chair.

Papilloma of the Larynx, with Symmetrical Papillomata on the Palate.

By ANDREW WYLIE, M.D.

THE patient, a male aged 40, a cab-driver, complains of hoarseness and, at times, dyspnœa. The hoarseness began eight months ago, gradually becoming worse, and latterly attacks of dyspnœa caused distress upon lying down. There is no dysphagia and no loss of weight. Upon examination there is a large subglottic papilloma in the anterior commissure of the larynx and two papillomatous growths on the edge of the soft palate, equal distances from the uvula. The exhibitor considers this a suitable case for the demonstration of the symmetry of the papillomata on the soft palate, and for the proof of their contagious nature. The case was first seen two weeks ago, and it is intended to remove the laryngeal papilloma by means of the direct method, which is especially applicable for growths of the larynx.

Dr. JOBSON HORNE inquired what might be meant by the "contagious nature" of papillomata on the soft palate. The growths were symmetrically placed on the free margin of the soft palate and close to the base of the uvula. Upon movement of the parts he observed no indication of contact of the growths. The symmetry of the situation of the growths was not usual, but it did not, in Dr. Horne's opinion, support the theory of their origin by contagion.

Dr. WYLIE, in reply, said that he thought the second papilloma had been caused by the soft palate rubbing against the original growth.

Removal of a Rapidly-growing soft Fibroma from Posterior Wall of Left Maxillary Antrum by a modified Rouge's Operation.

By HERBERT TILLEY, F.R.C.S.

H. B., AGED 48, complained of left nasal obstruction, increasing for twelve months. During past six months frequent nose-bleeding on that side, with discharges of glairy mucus. Examination showed posterior half of left nasal cavity completely filled by a dark-red, very soft, and easily bleeding mass of growth. Operation: Laryngotomy, plugging lower pharynx; incision under lip from right canine fossa to left malar process; division of anterior half of nasal septum from its lower attachment; turning up of soft parts; removal of left canine fossa and ascending process of superior maxillary; removal of growth and final replacement of soft parts, secured by a few interrupted sutures.

Specimen, with sections, shown.

DISCUSSION.

Dr. WATSON WILLIAMS said he saw the patient soon after the operation, and was struck by the rapid way in which he recovered from the operation, which gave free access to the growth, which of course it was very important to remove completely.

Mr. ROSE asked what was the point of attachment of the growth. He suggested that the Morbid Growths Committee should have the specimen for examination.

Dr. STCLAIR THOMSON said this was a modification of Rouge's operation, really Denker's. Mr. Tilley not only opened into the nose by Rouge's operation, but took away the ascending process of the superior maxilla, so as to enter the antrum at the same time. It was a most successful operation, owing to the absence of disfigurement, the rapidity with which the parts healed, and the fact that the septum was intact. Rhinologists should impress this operation on the general surgeon, who was a little too fond of taking away the upper jaw. If the attachment of an endo-nasal tumour was higher up, it could be easily reached by Moure's operation, and access obtained to the sphenoid and to the roof of the nose. There was another reason for not removing the upper jaw: those growths did not spring from the floor of the antrum, but from the ethmoidal region. The cribriform plate could be easily reached by that operation, or by a combination of Rouge's and Moure's operations.

The PRESIDENT (Dr. Dundas Grant) said he assumed it would have been impossible to remove the growth by way of the canine fossa alone, and he asked whether there was a large orifice leading from the antrum into the nose.

Mr. CHICHELE NOURSE asked how long it was since the operation was performed. In operations intended for the removal of growths in the upper part of the antrum, the great point was the removal of the ascending process of the superior maxilla, so as to gain free access. The plan adopted in the present case was very successful.

Mr. HERBERT TILLEY replied that he could not say exactly where the growth was attached. The patient came because of hæmorrhage, and the growth bled on the slightest provocation. It was a sessile growth, and apparently grew from the junction of the posterior wall of the antrum and the inner wall of the nose. When the attempt was made to remove it, it came away almost as easily as if it had been a loose body. It was important to cut through the wall of the septum at the attachment to the floor of the nose. The tracheotomy tube was put in, not only because it kept the anæsthetist out of the way, but because it permitted the mouth to be closed, so that the soft parts of the face could be turned upwards. In the case mentioned by Dr. William Hill, he thought necrosis of the hard palate would have occurred in any case, and was not due to the operation.

Papillomata of Larynx from a Boy aged 6, removed by Direct Method.

By HERBERT TILLEY, F.R.C.S.

THE patient has been operated on by me four times previously, but the growths have rapidly recurred. For several months he has worn a tracheotomy tube, and the granulations around this had become transformed into a hard, warty mass the size of a half-walnut. There is also a papillomatous growth on the posterior pharyngeal wall opposite the tip of the epiglottis. Suggestions for treatment are invited.

DISCUSSION.

Dr. WATSON WILLIAMS asked the President if he had not recommended salicylic acid in the treatment of such conditions; he would like to know how far his experience with it had been successful, and whether he would advise its use in this case.

Dr. JOBSON HORNE mentioned a case of papillomata of the larynx in a child aged 12 months, which was under his care years ago prior to the development

of the endolaryngeal treatment of those growths by the direct method. The child was wearing a tracheotomy tube, and had been doing so for some months previously, and the case was referred to Dr. Horne to remove the tube. The child was suffering from broncho-pneumonia at the time. As soon as possible Dr. Horne performed a thyrotomy and removed all the growths, cauterizing the site of origin. Subsequently the wound was completely closed and an intubation tube was inserted. The child made a good recovery, and speech was developed before leaving the hospital.

Dr. STCLAIR THOMSON said four times was too modest a frequency on which to operate for such a condition. One such case he had was operated upon at least sixteen times, and he thought the only thing to do was to go on "pegging away" at them. The conclusion of his case was that his patient finished up without a tube, and, from being practically voiceless, had a good voice.

Mr. MARK HOVELL said he had had a similar case, and after repeated operations the patient got well.

Dr. FITZGERALD POWELL said that cases such as these had been reported, in which a tracheotomy tube had been introduced and no attempt made to operate on the growths. The recurrent growth of the neoplasms was arrested and the patients got well after a time.

The PRESIDENT said he had had under care a girl aged 20 who had worn a tube from her fourth year. Her larynx was the seat of very large papillomatous growths, which had not disappeared, in spite of her having worn a tracheotomy tube for sixteen years. He (Dr. Grant) removed the growths and closed the tracheotomy fistula. A similar case, with equal lack of result from tracheotomy alone, was seen by Dr. Hunter Mackenzie, of Edinburgh, with similar persistence of the growths. Still, tracheotomy should be tried before thyrotomy was done. He had used salicylic acid for only small growths; he did not think anyone would be so sanguine as to expect it to have effect on huge warty growths. Recurrences after mechanical removal could be treated with salicylic acid, or the more modern ionization might be thought of.

Mr. WAGGETT asked Dr. Paterson if he could explain the reported frequency of these cases at Cardiff.

Dr. PATERSON, answering Mr. Waggett, said they did get such cases in good numbers, and had two or three on hand now. He could not explain their occurrence by any predisposing conditions in the district. They persevered with them by operating as many times as necessary. Three or four had cleared up altogether with that treatment. He was also in the habit of giving them a course of arsenic, as advocated by Körner, of Rostock.

Mr. HERBERT TILLEY, in reply, said that five years ago the boy was taken to a hospital as he had difficulty in breathing, and it was assumed he had diphtheria. So he was tracheotomized and put into the diphtheria ward, but a few days afterwards he was judged to be too well and comfortable for a patient

with diphtheria, and examination by the direct method showed the larynx to be full of papillomata. They had been operated upon several times, and on one occasion salicylic acid, 20 gr. to the ounce of absolute alcohol, was applied to the regions from which the warts had been removed. The patient then had a three or four weeks' course of arsenic. A fortnight ago there was an enormous number of papillomata filling the larynx to the tip of the epiglottis and extending down the trachea. He intended to persist with the operations as long as the growths recurred and the patient remained alive. The case to which Dr. Thomson referred was chloroformed twenty-one times, and when he saw him in the country some time ago he was acting as an efficient caddie and had a very good voice.

Specimen of the Face and Mouth of a Female Infant, showing arrest of Development of the Right Half of the Tongue, combined with a Cleft of the Soft Palate and a Palato-lingual Fold.

By A. R. TWEEDIE, F.R.C.S.

THE specimen was taken from a "wasting" child which was brought to the Nottingham Children's Hospital in October, 1909, as the parents found they were unable to feed it properly on account of a deformity in connexion with the mouth. The child was then eleven days old. An operation was suggested, but before it could be admitted for such treatment it died in a marasmic condition, aged three months. There is a complete congenital cleft of the whole soft palate, the left half of which lies in the position usually seen in such circumstances, but the right half is continuous with a membranous structure which, commencing opposite the posterior border of the hard palate, is attached continuously to the inner side of the right cheek and extends downwards and forwards to the floor of the mouth, where it terminates in front of the right side of the tip of the tongue. The specimen is shown as constituting a possible instance of the persistence on the right side of the lower half of the septum between the primitive stomodæum and foregut (with which the right side of the soft palate has become incorporated), which in its upper portion is sometimes represented by the condition known as congenital atresia of the posterior choanæ.

Pathological Report by A. Keith, M.D.—In fig. 1 the child's mouth shows the lips dissected away and the mouth opened. The wide cleft

in the soft palate is shown, but on the right side the uvula (*c*) and corresponding half of the palate are drawn downwards to form part of a palato-glossal fold. It is possible, as Mr. Tweedie suggests, that this fold is a remnant of the bucco-pharyngeal membrane which normally disappears in the third week of foetal life. Fig. 2 shows the appearance of the palatal cleft from behind: the left half (*a*) ends posteriorly in the uvula; below it a fold of mucous membrane is seen (*h*), which represents a large plica semilunaris. The right half of the palate is seen (fig. 2, *b*) pulled forwards into the abnormal palato-glossal fold. The attachments of this fold are best shown in fig. 1; anteriorly it appears to end at the tip of the tongue, but in reality in the tip of the

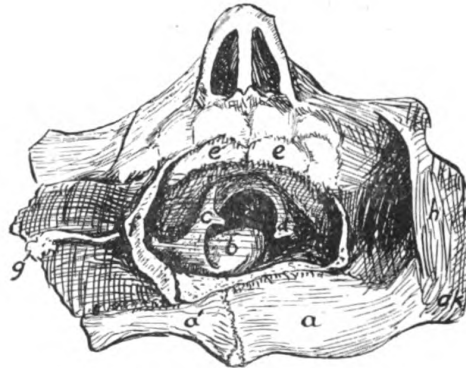


Fig. 1

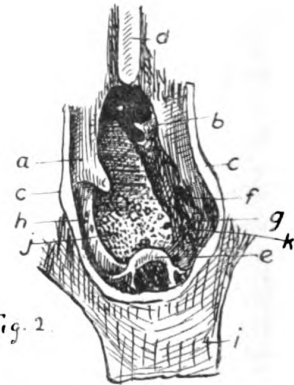


Fig. 2

FIG. 1.—Specimen from the front: *a*, *a'*, left and right (atrophied) halves of the mandible; *b*, left half of tongue; *c*, near uvula on the palato-glossal fold; *d*, left half of palate; *e*, *e'*, over upper median incisors; *f*, section of right cheek; *g*, parotid; *h*, masseter.

FIG. 2.—Specimen from behind: *a*, left half of palate; *b*, right half of palate and palato-glossal fold; *c*, wall of pharynx; *d*, septum of nose (cut); *e*, epiglottis; *f*, right Eustachian tube; *g*, right tonsil; *h*, plica semilunaris; *i*, pharynx; *j*, tongue; *k*, foramen cæcum.

left half of the tongue, for the fold is attached to the floor of the mouth, along the site which ought to be occupied by the right half of the tongue. The abnormal palato-glossal fold ends behind in the position usually occupied by the anterior pillar of the fauces. From the middle of the palato-glossal fold a ridge of mucous membrane passes outwards to the

right cheek (fig. 1, *f*). As already said, only the right half of the tongue is developed (see fig. 1, *b*, and fig. 2, *j*). The foramen cæcum (*k* in fig. 2) and the left circumvallate papillæ are present. Correlated with the above-mentioned anomalies are certain others affecting the parts developed out of the first or mandibular arch of the embryo. The right half of the jaw is arrested in its development; the left half is of a normal depth (10 mm.), whereas the right half of the jaw is only 5 mm. in depth. The body of the right half is 5 mm. shorter than the left. The submaxillary and sublingual glands were absent on the right side. In the Museum of the College of Surgeons there are three specimens which throw light on Mr. Tweedie's specimen. There is, first, one showing the floor of the mouth of a malformed child, in which a deep (5 mm.) paralingual fold of mucous membrane is situated on the floor of the mouth, over the site of the left submaxillary gland. It commences behind in the anterior pillar of the fauces and ends on the under surface of the tongue at the site of the plica fimbriata. Two other specimens show the paralingual fold, but with the fold there is a partial arrest in the development of the tongue and submaxillary and sublingual glands with an ossification of Meckel's cartilage, described by me in the current volume of the *Journal of Anatomy and Physiology*.¹ It is manifest that Mr. Tweedie's specimen is the result of a maldevelopment affecting the first or mandibular arch and the pharyngeal floor behind that arch. The musculature of the tongue is derived from behind the pharynx; it grows into mucous folds derived from the mandibular arch. In Mr. Tweedie's specimen the invasion of the musculature of the right half has not taken place, and the anterior part of the abnormal palato-glossal fold probably represents the mucous basis of the tongue. The condition is more than a mere arrest of muscular invasion, for the salivary glands which grow out in connexion with the primary lingual folds have also been arrested in their development, and the right half of the mandible is arrested in growth. The right parotid gland is normal. I have to thank Mr. Tweedie for presenting this remarkable specimen to the Museum of the Royal College of Surgeons of England.

DISCUSSION.

Dr. PATERSON said possibly the explanation was that the palatal process had got fused on the right side with the under surface of the tongue. It might be recalled that at one stage the tongue was clasped by the palatal processes.

¹ *Journ. Anat. and Physiol.*, 1910, xliv, p. 151.

102 Hastings: "*Bleeding Polypus*" of *Inferior Turbinate*

He did not think it had anything to do with the septum between the stomodæum and foregut, which disappeared very early, nor did he admit that congenital atresia of the posterior choanæ had reference to that structure.

Mr. TWEEDIE said he made the suggestion because it arose in the right plane from an embryological point of view. He had never seen a similar case. It was not fixed to the tongue, because during life the tongue could be freely moved behind it.

A Case of "*Bleeding Polypus*" of the Inferior Turbinate.

By SOMERVILLE HASTINGS, F.R.C.S.

ON November 8, 1909, a brass-finisher, aged 42, came to the Middlesex Hospital complaining of epistaxis of a month's duration. Every time he blew his nose he was troubled by bleeding from the

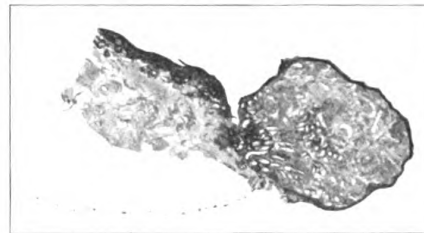


FIG. 1.

General view of section ($\times 6$).

right nostril, which lasted about a quarter of an hour. The bleeding was not severe and never came on spontaneously. When the nose was examined a pedunculated growth about the size of a pea was seen growing from the lower border of the right inferior turbinate not far from its anterior extremity. The tumour was purple in colour and smooth on the surface, and blood-clot was adhering to it at one point. Except that the mucous membrane on both sides of the nose was deeply congested—a fact easily accounted for by the alcoholic condition of the man—no other abnormality was noted. The small growth was snipped off with a piece of the mucous membrane covering the

inferior turbinate bone attached to it. When the patient returned to the hospital on November 21 the little wound had healed and there was no sign of any recurrence of the growth; and on December 30, in reply to a letter, the man stated that he was quite well.

A median longitudinal section of the tumour shows it to be of the nature of a soft angio-fibroma. The groundwork of the swelling is

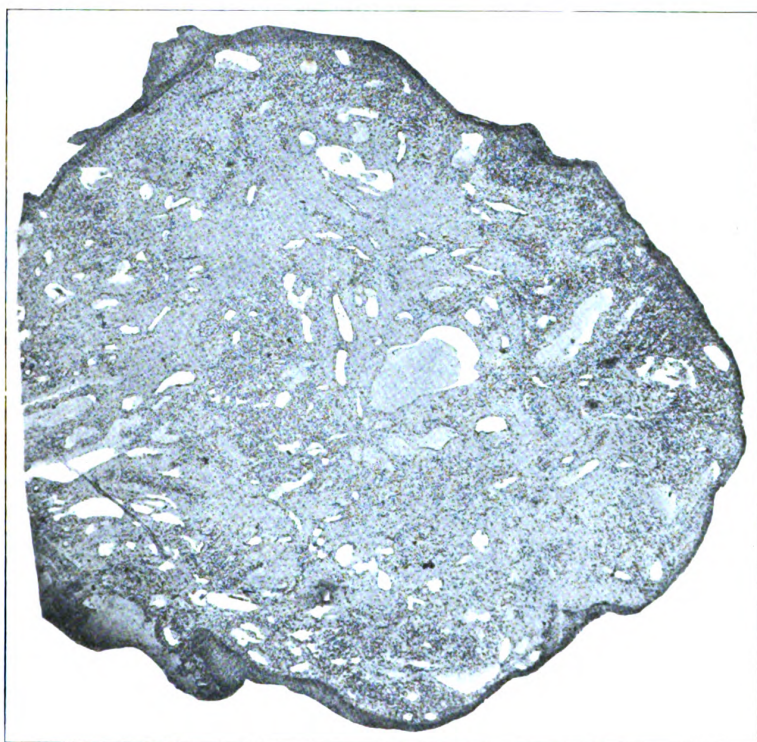


FIG. 2.

General view of tumour ($\times 30$).

composed of a fibrous matrix, in which are imbedded numerous oval connective-tissue cells of the endothelial type. A layer of squamous epithelium covers the tumour, and at one point, where ulceration is taking place, both tumour-substance and epithelium covering it are infiltrated with polymorphonuclear leucocytes. No plasma-cells (of

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Unna) are to be seen, nor are any mucous glands present in the tumour, though abundant in the adjoining mucous membrane. Scattered through the growth are many spaces lined with endothelium. Some of these contain blood; others, which are empty, may be lymphatics.



FIG 3.

Section showing attachment of tumour to inferior turbinate, and mucous glands in latter ($\times 55$).

As will readily be seen, the histological structure of the above tumour closely resembles that of "bleeding polypus of the septum," fully described in the papers by Dr. Pegler in the *Lancet*,¹ from which the

¹ *Lancet*, 1905, ii, pp. 1455, 1537.

following citations have been derived. Very few tumours of this structure appear to have been recorded growing from the inferior turbinate. Krieg¹ mentions an example which occurred in a woman aged 31; and Seifert and Kahn² describe the histological structure of these growths, and point out that this is quite different from that of mere hypertrophy of the mucous membrane. Schwager³ gives details of six cases and reviews the literature of the subject.

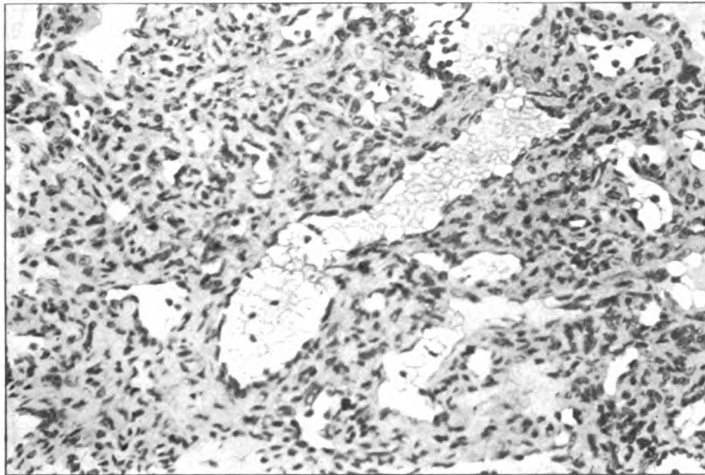


FIG. 4.

Section of tumour, showing endothelioid cells and vascular spaces ($\times 200$).

DISCUSSION.

Mr. F. A. ROSE said that if one used the term "bleeding polypus" to indicate tumours which were commonly found arising from the front of the septum, he would have doubt whether the tumour now shown was of the same class. Under the microscope it did not seem to resemble any of three specimens he possessed which had been removed from the septum. All his specimens showed large angiomatous spaces.

¹ "Atlas of Diseases of the Nose" (Eng. transl.), Stuttg., 1901.

² "Atlas der Histopathologie der Nase," Weisb., 1895.

³ *Archiv f. Laryng. u. Rhin.*, Berl., 1894, i, p. 105.

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The PRESIDENT said that some time ago he was called to see a lady, aged 63, who had had some obstruction in her right nostril for only a few weeks. She had had spontaneous bleeding, which had been associated with a thick discharge, and her health was suffering. The obstructing body looked like a polypus, with an angry-looking, reddish surface, which bled readily. It caused bulging of the nose below the lower margin of the nasal bone, where there was a boggy area which was tender on pressure. There were no enlarged glands, but from the appearance of the growth he thought it most probably malignant. He removed it. It appeared to grow from the upper and anterior part of the inferior turbinate body, and the pathological report was that it was an angio-myxomatous tumour. Mr. Butlin also saw it, and thought it looked malignant. He would bring the section before the Society. The pathologist reported it to be angio-myxomatous in structure.

Dr. PEGLER said Mr. Hastings was fortunate in having shown the first example of discrete angioma which, so far as the speaker knew, had been seen growing from the inferior turbinal in this country. The section was best appreciated when viewed with a magnifying glass. Certain other cases, but growing from the triangular cartilage of the septum, showing the relation of the angioma to the base of the attachment, were Dr. Kelson's and was one of Dr. Furniss Potter's; in all these one saw the actual starting point of the angiomatous growth, but none of the specimens yet seen had helped much in deciding the ever-green question whether we were dealing with a vascular change in granulomatous tissue or with a purely angiomatous formation. With regard to Dr. Rose's question as to diagnosis, Dr. Pegler said that if that gentleman would examine the large collection of examples in the Society's cabinet in the library, he would have a better opportunity of studying the great variation that these angiomas exhibited. The present one agreed in many respects with Dr. Scanes Spicer's soft fibro-angioma of the septum; the patient in that case was shown at one of the meetings of the old Society with the growth *in situ*. He would emphasize especially the presence of blood spaces in the pedicle and body of the growths, of areas of what he had designated fibro-angiomatous meshwork, in the trabeculae of which were scattered, as elsewhere in the growth, abundance of the characteristic "endothelioid" cells; also the negative facts of complete absence of non-striped muscular fibre around the vascular spaces and of mucous glands, both these elements being well represented in the turbinal part of the section. "Bleeding polypus" was by no means confined to the septum, nor to any part of it.

Mr. HASTINGS replied that he did not think the tumour could be a mere hypertrophy of the inferior turbinate because it was definitely pedunculated, and the inferior turbinate looked otherwise healthy, and so did the turbinate on the other side. Moreover, the tumour contained no glands, and its vascular spaces, unlike those of the inferior turbinate, were entirely devoid of muscular wall. His own specimen corresponded very well with the diagrams of some of the specimens of "bleeding polypus" of the septum he found described in papers on the subject.

**Larynx, Four Rings of Trachea, and part of Thyroid Gland
and Gullet removed during an act of Suicide.**

By E. A. PETERS, M.D.

THIS specimen was given me by Dr. Clark, of Rickmansworth. A painter, aged 29, subject to hallucinations and depression after bouts of alcoholism, cut his throat when sober at 5 a.m. He made a transverse cut down to the spine and two or three vertical cuts, one of which opened the larynx accurately in the middle line. The suicide seized the obstructing larynx, cut away the adherent gullet and trachea at the fifth ring, and threw the fragment exhibited into the garden; he walked 200 yards, to collapse outside a friend's house, where he was discovered. Dr. Evans, who saw him first, found no bleeding vessels. The two doctors found it impossible to bring the trachea to the skin. Death from lung suffocation followed at 10 a.m.

" Singer's Node " on Left Cord

By E. A. PETERS, M.D.

H. S., AGED 41, foreman fitter, lost his voice three months ago. It has improved somewhat lately, but his voice is still impaired. The small nodule, less than $\frac{1}{8}$ in. in diameter, is white and sessile, of the shape of a split pea. It is attached to the under surface and margin of the cord at the juncture of the anterior and middle third. What is the best method to remove this nodule?

DISCUSSION.

Mr. TILLEY suggested that the galvano-cautery should be used by the direct method. He had recently treated a case by that method, and it left nothing to be desired.

Dr. MILLIGAN suggested that attention should be paid to his nasopharynx, and that he should have vocal rest. Fifteen years ago he applied the galvano-cautery by the indirect method in the case of a young lady, and with very great care, and for a time there was an absolutely good result; then a little cicatricial

contraction took place, the cord became bowed, and the singing voice was permanently interfered with. He was averse to the use of the cautery in the treatment of small nodes, as it was quite impossible, however carefully it might be applied, to say with any certainty how much cicatricial contraction might take place.

Dr. FITZGERALD POWELL did not agree that the growth on the vocal cord in this case was a "singer's node." He would be inclined to regard it from its appearance and position as a fibroma, or a form of pachydermia. With regard to the treatment, he thought the application of a guarded galvano-cautery point would destroy it most effectively. He was afraid there was a tendency to advocate the use of the direct method to too great an extent, to the exclusion of the indirect method. In the case of true singer's nodes, he did not think anyone should attempt to interfere with the cords of a vocalist, as operative procedure might destroy the voice, and, in most cases, prolonged absolute rest of the voice would effect a cure.

Mr. BARWELL said he hoped the Section would not fall into the trap of discussing the relative advantages of the direct and indirect methods.

Mr. HORSFORD said that as there were cases in which the direct method was either disagreeable or unsuitable, he wished to remind the Section of the method of the epiglottic suture and the instrument devised for the purpose which he introduced some time ago.

The PRESIDENT said he had constantly hesitated to apply the galvano-cautery to those nodules. It was surprising to what extent the cases would recover even if the nodules were left there.

Dr. STCLAIR THOMSON said manipulations could be made by the indirect as well as by the direct method. Professor von Eicken removed nodules from the vocal cords by the indirect method in Killian's clinique. It was easier, quicker, and more pleasant for the patient. He hoped the present case would not be recorded as a singer's nodule; it was not in the classical position; it was not on the edge of the cord; and there was no affection of the opposite cord.

Dr. WYLIE agreed with Dr. Powell that it was not a singer's node, and he thought that it could easily be removed by Dr. Grant's laryngeal forceps and the upper respiratory tract attended to. He considered that there was a want of movement of the vocal cords, that there would be a difficulty in employing the galvano-cautery, and it would not be so satisfactory.

Dr. PETERS replied that it was not a singer's node in the true sense, but it was equivalent to such. It started at the edge of the cord, and now it had diminished in size, and did not materially interfere with the voice. It seemed to be the result of attrition in a non-singing voice. It was under the cord, and therefore easy to manipulate, and he hoped to remove it by the galvano-cautery by the indirect method. He had not had bad results such as Dr. Milligan pointed out. The man refused treatment by rest, as he was a foreman fitter, and his voice was used a good deal at his work.

Tuberculous Perichondritis of Cricoid and Arytænoid Cartilages.

By HAROLD BARWELL, F.R.C.S.

THE patient, a man aged 64, was admitted to hospital under the care of my colleague, Dr. Cyril Ogle, with bronchitis, dilated heart, and anasarca. He had had some difficulty in swallowing for twelve months, and dyspnœa for four weeks. Syphilis many years ago; no history of tuberculosis. The exhibitor examined him on admission and found marked obstructive dyspnœa. A large, irregular mass occupied the left half of the larynx, the left arytænoid was fixed in the middle line, there was deficient abduction of the right cord, and a rounded swelling at the back of the cricoid plate projecting into the pharynx. It was thought to be a case of malignant disease, and tracheotomy was advised and performed the next day under local anæsthesia. The patient died ten days later from cardiac failure. Post mortem, Dr. Trevor found the cricoid and left arytænoid cartilages exposed and necrotic, but no tuberculous ulceration or infiltration of the superficial parts of the larynx. There was a quiescent fibro-caseous nodule in each lung. Dr. Slater has found tubercle bacilli in the sub-perichondrial necrotic tissue, and has inoculated a guinea-pig, but the result is not yet to hand (March 23). The inoculated animal has now been killed and shows advanced tuberculosis.

Perichondritis of Thyroid Cartilage of Unknown Origin.

By HAROLD BARWELL, F.R.C.S.

THE patient, a German waiter aged 47, was admitted on December 31, 1909, under my colleague, Dr. Collier. Dysphagia began five days before, and attacks of dyspnœa, with stridor, came on three days after admission; there were four such attacks on December 31, before admission. No previous illness; syphilis denied. There was some dyspnœa on that night, but not so serious as to demand tracheotomy, after which the difficulty in breathing ceased and the patient seemed convalescent. On January 7 the exhibitor found swelling of the right arytæno-epiglottidean fold and arytænoid, which was fixed

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in abduction, and diagnosed perichondritis. After this, though the patient did not complain of dysphagia, he refused food, and on January 11, 1910, died suddenly while asleep. Post mortem, Dr. Trevor found perichondritis of the right ala of the thyroid cartilage, which was bare on its inner aspect. An abscess ramifying between the gullet and the air-passage had ruptured into the pyriform fossa, and the contents had been aspirated during sleep.

Mr. BARWELL said that the swelling over the arytaenoid was only slight, and that the appearance in no way suggested the presence of an abscess. The ætiology of many of these cases was very obscure, and in the present instance no explanation was forthcoming.

Chronic Suppuration of the left Frontal Sinus, with Displacement of the left Eyeball and Diplopia ; Operation ; Recovery.

By CHICHELE NOURSE, F.R.C.S.Ed.

THE patient, a man aged 36, was referred to the author by Mr. de Gruyther in May, 1909, on account of proptosis and displacement outwards and downwards of the left eye, which had come on some days before ; and diplopia, which had troubled him for five months. There was considerable swelling of the frontal region on both sides, particularly the left, and the inner part of the roof of the left orbit was pushed downwards. No pain had been complained of at any time, there was no marked tenderness, and no fluctuation could be detected. The swelling was clearly due to expansion of the bony walls of the left frontal sinus. On transillumination both frontal sinuses and both antra were opaque. An X-ray photograph, kindly taken by Dr. Ironside Bruce, shows a marked shadow above the left orbit. Both maxillary antra contained pus.

The patient stated that he had been troubled with a discharge from the left nostril for ten years, and had occasionally noticed subjective fœtor. At the time of examination the left nasal fossa was free from pus, but the middle meatus contained some polypoid tissue of a doubtful appearance. Dr. Wyatt Wingrave's report of a specimen of this material was that it consisted of gland tissue, infiltrated with large mesoblastic cells of the endothelial type. The Ogston-Luc operation was performed

on May 21. An incision was made through the brow from $\frac{3}{4}$ in. to the right of the middle line as far as the junction of the outer and middle thirds of the left eyebrow, where it was carried a little way upwards and outwards. The bone was rough on the surface, and vascular; it bulged considerably, especially over the left frontal sinus; and at one spot it was thinned and bluish grey in colour. On making an opening through the bone, offensive pus immediately welled up, and on enlarging the opening the exposed lining of the sinus presented as a dark-red pulsating mass. The cavity contained a large quantity of fœtid pus, enclosed in the vascular and polypoid lining membrane. The bony wall of the sinus was uneven; at one point in the superior wall the dura was exposed to the extent of a threepenny-piece, and at another place the orbital wall was defective to about a similar extent. The septum between the two sinuses was situated a little to the right of the middle line, and was intact. The left frontal sinus formed a cavity of considerable depth and height; it measured $2\frac{1}{2}$ in. outwards from the septum. The fronto-nasal canal was large enough to admit the little finger. The whole contents of the sinus, including the lining, were removed, and solution of zinc chloride applied to the walls. A drainage-tube, sufficiently large to fit tightly (about No. 40 French gauge), was then passed down the infundibulum, and left projecting from the nostril. It was left in place for fourteen days. The cavity was lightly packed with gauze for twenty-four hours, and the wound closed with sutures, except at the inner end. For two days there was considerable œdema of the forehead and upper eyelid, but no further trouble occurred.

The operation wound had completely healed in ten days, but re-opened again in the middle line some weeks later, and did not finally close until November 19. The diplopia disappeared sixteen days after the operation, though the left eye remained distinctly at a lower level than the right for some weeks. The patient's general condition improved greatly, and he is now well, with the exception of slight suppuration in both maxillary antra, which is not quite cured.

Dr. Wyatt Wingrave reported that the pus from the frontal sinus was swarming with small Gram-negative diplococci, and also contained clumps of *Bacillus pyocyaneus fœtidus*.

Mr. NOURSE, in reply to the President, added that the swelling was bony, evidently due to the expansion of the bony wall of the left frontal sinus. The dura was exposed at one spot in the sinus. In regard to radiograms, Dr. Ironside Bruce said that when both sinuses were opaque in the front view, it was advisable to get lateral views of the sinuses as well, because the opacity might be due to non-existence of the sinus.

A Case of Tuberculous Disease of the Epiglottis.

By W. H. KELSON, M.D.

MAN, aged 36, commercial traveller. No history of syphilis. Mother died of phthisis. For four months has complained of cough and pain on swallowing. No tubercle bacilli have been found in the very scanty sputum, but crepitations are to be heard at both apices. Epiglottis presents a curiously distorted and ulcerated appearance.

Complete Inspiratory Stenosis of the Larynx, in a Man. Case for Diagnosis.

By STCLAIR THOMSON, M.D.

THIS man has been invalided from the Army in India on account of present condition. He states that twelve months ago his voice began to go, and the Army report shows that he had an ulcerated patch below the right vocal cord. He was admitted to hospital in India on February 11, 1909, with almost complete aphonia. No tubercle bacilli were found, and he was treated with Hg and KI, with no improvement. On August 6 he suddenly became cyanosed, and tracheotomy had to be performed. Admitted to King's College Hospital a few weeks ago, it was seen that a laryngotomy, and not a tracheotomy, had been carried out. He was wearing a small-sized tracheotomy-tube. There is no inspiration through the glottis, but there is enough expiration to enable him to talk in a rough whisper. The larynx is occluded by smooth, red swelling of both ventricular bands, so that they are in complete apposition. There is no ulceration, but on phonation a fleeting glance of some greyish mass is obtained in the region of the cords. The larynx is generally bathed in pus; from the outside it is felt to be enlarged, but freely movable. At first there was marked tenderness on the left side. There is no history of syphilis, but there is a scar on the penis. The family history is good. The patient lost 2 st. in weight, but of this he has recovered 9 lb. The sputum and discharge have been examined for tubercle bacilli, with negative result. There are no physical signs in the chest. The temperature is normal. The Wassermann and von Pirquet

reactions are negative. There are no enlarged glands. The patient swallows easily, and appears in fair health and good spirits. He has been examined by the Röntgen rays, but no metallic foreign body was disclosed. Examination by direct laryngoscopy did not reveal anything. The first step in treatment was to perform a genuine, median tracheotomy. It is interesting to compare the situation of the cannula at present with the scar of where it was before. Patient has been put on inunctions without decided improvement. The case is evidently not one of malignant disease or tubercle. There is, doubtless, perichondritis going on. The probability is that, after all, the condition is syphilitic, or due to some foreign body impacted in the ventricles of the larynx. Opinions are invited.

DISCUSSION.

Dr. PATERSON said he had had a case for the last year, in a collier, in whom there was no history of syphilis, and it had proceeded in much the same way, the interior of the larynx being filled up with swelling. As the man had a suffocative attack, the doctor in the country did laryngotomy. He (Dr. Paterson) did a low tracheotomy, which relieved him to some extent. But the swelling was very variable in extent: sometimes it seemed almost well, and at present some air passed through the larynx. He had not done a fissure. But Dr. Thomson's case was more advanced, and would perhaps require something further to be done.

Dr. MILLIGAN suggested that pus from the larynx should be collected with a curved pipette, that it should be bacteriologically examined, and a vaccine made from the predominating organism. The purulent condition present might be the result of some local infection, the result of previous disease—e.g., perichondritis.

Dr. FITZGERALD POWELL said this was a most interesting condition, but it was not at all easy to say what the condition was due to. He thought it was a case of perichondritis of the larynx, and probably due to syphilis. The grey mass alluded to by Dr. StClair Thomson was probably a portion of necrosed cartilage. He would like to know why Dr. Thomson suggested the possibility of a foreign body in the larynx as a cause of the trouble, and if, when he performed the tracheotomy, he had examined the larynx through the tracheotomy opening for the presence of a foreign body. The condition at present was too active to carry out any operative measures, but when the case had quieted down, and laryngeal stenosis remained, a good result might be obtained by removal of half the larynx. He had done this in a somewhat similar case, and the man recovered with a good voice.

Dr. THOMSON, in reply, said he spoke of foreign body because in a case of Mr. Lake's, some years ago, everyone was puzzled, and later on a spicule of

bone was removed. He would adopt Dr. Milligan's suggestion. They had examined for tubercle bacilli, but not for a special organism. The direct method showed less than the indirect. Nothing more was seen than a greyish something on the vocal cord. He was hoping to hear someone suggest laryngofissure. He agreed with Dr. Powell that at the tracheotomy he ought to have examined from below; but he did not think until afterwards of the possibility of foreign body.

Unilateral Pansinusitis in a Man aged 21; shown Six Weeks after Operation, when all Four Cavities were Operated on at One Sitting.

By STCLAIR THOMSON, M.D.

IT was intended to exhibit this patient at the last meeting—a fortnight after operation—to show how speedy is the recovery in a successful case. Slight temporary indisposition prevented this. A radiograph shows how extensive was the orbito-ethmoidal cell in this case. It extended far back in the roof of the orbit, resembling very much the one in the dried skull which is exhibited at the same time. With the exception of a little crust in the ethmoid region, all suppuration has ceased. Before the operation was undertaken, the frontal sinus was washed out on several occasions and the antrum was punctured and washed out three times. No relief followed, and the patient asked for the radical operation on account of the persistent headache. This has quite disappeared. There is no diplopia.

Cast of Upper Jaw from a Case of Congenital Occlusion of the Posterior Naris.

By D. R. PATERSON, M.D.

THE patient, a woman aged 41, did not complain of difficulty in breathing until two years ago. The right nasal cavity, after being emptied of mucus, was seen to be completely closed posteriorly, an excellent view being obtained as there was no septal deviation to interfere. The nasal floor on the right side was apparently lower than the left. There was no asymmetry of the face and no appreciable

difference noted on inspection in the two halves of the upper jaw, but the cast shows the right half to be actually narrower and the arch of the palate slightly higher on that side.

DISCUSSION.

Mr. ROSE asked whether the hearing was affected on the obstructed side.

Mr. CLAYTON FOX asked whether there was any mental deficiency, also whether the nasal fossa on the affected side was smaller than the other.

Dr. PATERSON replied that it was difficult to perforate the septum freely behind, because the inner edge of the Eustachian tube came so far towards the middle line that it encroached on the new opening; nevertheless the hearing was normal. There was no mental deficiency in the patient. The obstruction in the right nostril was probably not noticed, as free breathing was possible through the left side, until it became encroached upon by an enlarged turbinal. The difference in size of the two nasal cavities was slight.

Atresia of the Anterior Naris.

By NORMAN PATTERSON, F.R.C.S.

FEMALE, aged 23. The right anterior naris is closed by a union between the skin covering the anterior part of the nasal septum and that lining the ala. At the upper part of the union there is a small pocket into which a probe can be passed for about $\frac{1}{4}$ in. The tip of the nose is slightly deflected to the left. The cartilaginous septum is markedly bulged over to the right. The lateral incisors are rotated, their anterior surfaces looking forwards and inwards. The palate is not unusually high. There is nothing of note in the post-nasal space. Vague history of an injury to nose caused by a swing accident when a child. Opinions are solicited as regards the best form of treatment.

DISCUSSION.

Mr. CLAYTON FOX said it seemed to be a cicatricial web formation, probably the result of congenital syphilis; although the upper central incisors were not typical Hutchinson's teeth, she had pegged canines and dome-shaped molars. The web had the appearance of cicatricial tissue. Against it being a congenital web was the fact that it was more marked than such webs in that position; the developmental web was usually crescentic and an exaggeration of the normal semilunar fold found at the limen vestibuli. In this case the vestibule was closed.

Mr. HERBERT TILLEY said that when the patient was told to force air down the side of the scar, it would bulge forwards a little. Hence, if the scar tissue were perforated, and a partial submucous resection performed, the patient would get a perfect nostril.

Mr. NORMAN PATTERSON replied that possibly it might be due to congenital syphilis, but it might be developmental. He got Dr. Arthur Keith to see the case. Dr. Keith said that this region of the nose was closed by a plug of epithelium from the third to the seventh month of intra-uterine life, which became invaded by mesoblast and organized. He proposed to do a modified Killian's operation.

Case of Asthma ; Improvement after Nasal Treatment.

By DAN MCKENZIE, M.D.

THE patient is a male, aged 34, who has suffered from asthma since childhood. Both middle turbinals, which were enlarged—the right being polypoid and cellular—were snared in September and October, 1907. The septum was resected under cocaine in February, 1908. He states that he has not had a paroxysm now for seven months.

DISCUSSION.

Dr. SCANES SPICER said this was a very satisfactory illustration of the effect of suitable and adequate operation on the nose in asthma. In these cases substantial progress had been made since the submucous resection operation had been perfected. In this case, both middle turbinals were first removed, but the symptoms continued. Dr. McKenzie had rightly judged there was pressure or tension of nasal nerves, due to the bony or cartilaginous part of the septum being bent or pressed against the outer walls of the nasal cavities. This might be inferred from the favourable result of the submucous resection. No other operation on the septum in the cases he meant—i.e., very high up and anterior deflections—could effect the object desired short of free ablation of the septum (which personally he had only done on two occasions as a last resort) before the modern submucous resection methods were perfected.

Dr. PEGLER expressed disagreement with Dr. Spicer. In former years, if they did not do submucous resection, they had other methods which arrived at the same result, and he for one was quite satisfied with his early cases. He fully recognized the benefit these patients derived both from clearing up obstruction to breathing and in a certain proportion from cauterization of the septum, but he thought the *modus operandi* of these measures was still obscure.

Mr. CLAYTON FOX asked whether the application of adrenalin or other vascular constrictants had been tried; in these cases it often afforded a clue as to the utility of the galvano-cautery. If there were bony or other hard enlargements present causing pressure, they should be removed.

Dr. H. J. DAVIS said if adrenalin was given, it was much better to inject it subcutaneously, as the result was then more rapid. If an asthmatic had disease of the nose and polypi, and enlarged turbinals, unless one cleared the whole of that away the patient would not be cured of the asthma. But many asthmatics had normal noses, and if only the septum of those were cauterized they got relief. He had five such cases now, and that was their only treatment. Unless the cautery cut, as pointed out by Francis, was made in the right spot, it was of no use as an alleviation.

The PRESIDENT desired to congratulate Dr. McKenzie on the patient's freedom from attack for seven months. He hoped more would be heard of the case. Many members would have had cases in which they were disappointed by recurrence, even after months of freedom. Others had got well after even less operative activity than Dr. McKenzie had practised in the present instance. But the time had passed when anyone could deprecate the idea of treating asthma by means of operations on the nose. The Laryngological Society some years ago appeared to have expressed an opinion unfavourable to the treatment now being discussed. Since that date there had been the scientific investigations of Dixon, as well as Francis's clinical observations, and, though Francis's results were not found in every case, all must have had cases which answered to the treatment. Possibly the subject would come up again.

Dr. MCKENZIE, in reply, said he brought the case with the idea that the Section might focus present opinion on the matter. The asthma had been relieved beyond his expectation. His experience was that improvement was less marked with the cautery. Previous to his operations, which were necessary on account of the nasal obstruction, the patient could get no sleep until 4 a.m., but now he never had a bad night, and slept soundly from the time he went to bed until he got up.

Case of Œsophageal Stenosis in a Man aged 53.

By R. H. SCANES SPICER, M.D.

SYMPTOMS of dysphagia of three months' duration. Demonstration with Brünings' œsophagoscope. Radiogram by Dr. Harrison Orton, showing bismuth porridge shadow 3 in. to 4 in. long in stenosed part between heart and vertebral column. Patient is kyphotic and a belly-breather.

It is suggested that such a subject during muscular exertion, even doing light work such as a storekeeper, on straightening his spine is likely to subject his gullet to undue stress (pressure between heart, &c., and spine), strain (pull owing to descent of diaphragm and abdominal viscera), and friction (rub between moving viscera and spinal column). Such excessive intrinsic mechanical forces must impinge mainly about three places: First, the area of excursion of the cricoid downwards; secondly, behind the tracheal bifurcation and left bronchus; thirdly, about that portion of the oesophagus which is in contact with its moving foramen in the diaphragm. The brunt of the conflict has fallen in this case on the second of the above sites, and the diagnosis does not admit of much doubt.

Dr. SCANES SPICER added that he had not examined this case with the endoscope at all when the notice was sent in for the programme. He had since done so. The stenosed portion was 31-36 cm. from the incisor teeth. The lumen was narrowed generally, and there was a hard, fibrous, irregular spiral band running down and round the gullet. At places, little white nodules, small cauliflower patches, a dilated venous sac, and some small superficial ulcerations were to be made out. Two small bits were picked off for examination. He had had a report from Dr. Spilsbury that both were squamous epitheliomata with a fibrous stroma. He wished to hear the opinion of those members who had actually treated gullet cancers with radium, whether they considered this a favourable case for treatment by radiant energy, and especially by radium. He could get the second largest Brünings' extension tube through the stricture, so it would seem in that respect to be a favourable case for the conjunction of the radium tube with the actual site of cancer. It was not a soft, rapidly growing cauliflower condition, which would break down quickly and lead to perforation. The patient had been a drummer boy in the Army from the age of 13 to 22, and had then been invalided out of the Army for "palpitation" and "nervousness." The site of the present stricture was behind the heart. Was there possibly a connexion between the old palpitation and the present stenosis? The concurrence of oesophageal cancer and aortic aneurism had been previously recorded.

Dr. FINZI said the patient had a growth in the oesophagus which was squamous-celled epithelioma, and that was clearly visible in the radiogram. It would be easy to get the radium to it, but squamous growth was not a favourable kind of tumour for radium. If it had been carcinoma it would have been a favourable case, because it was limited, and it moved with respiration. But as he could not, perhaps, be benefited in any other way, it would be as well to try radium.

A Case of Intranasal Disease (Obstruction, Pressure, and Ethmoidal Suppuration), associated with aggravated, lifelong Stammering.

By R. H. SCANES SPICER, M.D.

THE intranasal disease was cured by operation in September, 1909. (Partial reduction of both inferior turbinals. Resection of right middle turbinal. Curettement of ethmoidal cells.) The patient is shown chiefly to illustrate the psychico-postural-respiratory method by which he can instantaneously arrest his attacks of stammering, and by which he is gradually being trained to conquer the habit permanently.

Laryngeal Case for Diagnosis: Abductor Paralysis right side, Abductor Paresis left side.

By H. J. DAVIS, M.D.

A MAN, aged 76. Left vocal cord in cadaveric position; the right is now becoming similarly affected: both ventricular bands partially obscure the cords. There is slight pain on swallowing. An ulcer, or rather its margin, was visible three months ago, extending behind the arytænoids; this has vanished under iodides. A piece was removed by direct laryngoscopy, and the pathological report was negative. In spite of this, the exhibitor looks upon the case as malignant. Both cords will, later, become fixed in the mid-line, and both recurrent laryngeal nerves are evidently implicated in an œsophageal ulcer.

Dr. DAVIS added that most members did not think it was malignant, but he felt certain that it was; there was double abductor paralysis, and he believed there was malignant disease of the œsophagus invading both laryngeal nerves. One side had become paralysed while the patient was under observation. He used to have an ulcer visible above the arytænoids, but that certainly had disappeared with iodide treatment. He was sure, owing to his dysphagia, there was obstruction lower down, and that both recurrent laryngeal nerves were implicated in the growth.

Dr. WILLIAM HILL showed :—

(1) Carcinoma of gullet in a man aged 62. Demonstration of radium apparatus and its insertion in gullet by means of a special œsophageal tube designed by exhibitor.

(2) Skiagrams of cases of carcinoma of gullet, with radium tubes in the various thicknesses of screens photographed *in situ*.

(3) Traumatic cicatricial stenosis of larynx in a child aged 6, under treatment by a rubber intubation tube.

Laryngological Section.

April 1, 1910.

Dr. J. DUNDAS GRANT, President of the Section, in the Chair.

A Case of Healed Tuberculous Laryngitis, treated by Galvano-Puncture.

By HERBERT TILLEY, F.R.C.S.

THE patient, a woman aged 27, complained of "hoarseness," and was under sanatorium treatment for pulmonary tuberculosis. The left ventricular band was swollen, so that the corresponding vocal cord was invisible. Four deep punctures with the galvano-cautery had brought about the present satisfactory condition.

DISCUSSION.

Dr. JOBSON HORNE desired in the first place to congratulate Dr. Tilley upon the result achieved in the case, and next to speak generally and irrespective of the case before them. A remark that had been made by a wise man with reference to the excellent results obtained by means of the submucous resection operation of the nasal septum—the best results were yielded by those cases which stood most in need of the operation—was equally applicable to the results obtained by the galvano-cautery treatment of laryngeal tuberculosis. There was a factor in these cases overlooked when results were satisfactory, and fallen back upon when not satisfactory, and that factor was Providence. He (Dr. Horne) was not disparaging the treatment by means of the galvano-cautery; on the contrary, he was one of the first to urge it. He desired to emphasize the suitable and the unsuitable cases. The latter were those running an acute course, the former were those with a natural tendency to heal.

Dr. DAN MCKENZIE showed the meeting some galvano-cautery points which he had found convenient in the application of galvano-cautery puncture to the larynx. Their peculiar feature lay in the fact that the platinum terminal was set at right angles to the shaft, so that it was possible to apply the point with accuracy and precision to the selected spot. Four varieties were made in all, for application to the anterior, posterior, and lateral walls of the larynx. They could be used either by the direct or by the indirect method.

Dr. SCANES SPICER said the question was as to whether there were laryngeal symptoms. He supposed that it was for hoarseness in the present case that Mr. Tilley did the galvano-puncture. The results were very satisfactory, but he noticed some redness on the right cord of the opposite side. The patient was now husky, but he (Dr. Spicer) could not make out the cause of the huskiness. There was no evidence of tuberculosis in the larynx at present.

Dr. DONELAN said Mr. Tilley was to be congratulated on the excellent result. He (the speaker) had had much to do with cases of the same kind in the last few years. Two particular cases had been punctured fifteen or sixteen times each. A great improvement had been effected; he had shown one case here. He thought he could have eradicated the disease had he used the cautery less timidly. Recently a more decided use at longer intervals had produced excellent results. Operators had not hitherto been bold enough in employing the cautery early so as at once to eradicate any local disease which might be present; they seemed afraid of the reaction which sometimes took place after interlaryngeal interference; but in tuberculous cases there appeared to be less tendency to reaction after irritation than in a state of health. He had recently applied a large cautery twice a week for three weeks in a severely ulcerated epiglottis, so far with good results.

Dr. FITZGERALD POWELL asked whether the four deep punctures with the galvano-cautery were made at one sitting. With regard to "reaction," in some of his cases he punctured three or four weeks consecutively, and there was considerable reaction, probably because he did it too often. He would like to hear the experience of others as to how often it should be done.

Mr. HORSFORD asked whether in such cases Mr. Tilley allowed his treatment to be influenced by the lung condition. If the laryngeal condition alone was suitable for a certain treatment, did the extensiveness of the lung condition influence the treatment of the larynx?

Dr. W. HILL asked whether Mr. Tilley had had much experience of the galvano-cautery (not galvano-puncture) in conditions in which there was not marked tumefaction, but a slight, irregular ulcerated condition of the vocal cords. He had recently tried it in a case, but there was much reaction, and he could not trace much benefit; perhaps because he had not persevered sufficiently.

The PRESIDENT (Dr. Dundas Grant) said the old question of *post hoc* and *propter hoc* inevitably cropped up in regard to any new remedy; but those who could remember cases where the circumstances had been otherwise the same, but in which the present treatment had not been tried, would realize that the results obtained by its use were much better than those formerly obtained, and in very many cases much better than it seemed reasonable to expect. He certainly was in favour of galvano-caustic puncture rather than superficial burning, because there was a greater sclerotic effect. Superficial cauterization of tuberculous ulcers seemed to be beneficial, especially in relieving pain. The absence of reaction after galvano-caustic puncture was almost a revelation. He had often been deterred from using the galvano-cautery when the patient had been going rapidly down hill because he feared to bring the treatment into discredit, but he was not quite sure that even in those cases its use might not have been beneficial.

Mr. HERBERT TILLEY, in reply, said the reason he did not state the history on the printed paper was that he did not want to write a long history, and probably if he had many would not have read it. Last spring the patient was sent with the history that she had been in the Midhurst Sanatorium six months, and that during the last two months, in spite of the open-air and "silence" treatment, her voice had become progressively worse, and she had begun to complain of pain in her throat. He believed she had had a few applications of lactic acid to the throat. When he examined her she was hoarse, but on the present occasion her voice was particularly good. He made a drawing of the laryngeal condition when he first saw it, and had noted that the anterior two-thirds of the left cord was invisible owing to the swelling of the ventricular band. It was also stated in her report that she had some tubercle bacilli in her sputum, but otherwise her general health had improved under the sanatorium treatment. Those facts cut the ground from Dr. Jobson Horne's criticism, because that speaker argued that if the case had been left alone she would probably have got well. He made two punctures at the first sitting, burying the point of the needle $\frac{1}{2}$ in. into the ventricular band. A fortnight later she came up again, and he punctured the band again, and a fortnight later repeated the process. She was still persisting with the sanatorium treatment at her home, and the total result was her present improved condition and her return to work as a teacher, with restoration of voice. Therefore he thought the local treatment should be given a large share of credit in the improved laryngeal condition. In answer to Mr. Horsford, if such a patient came with a report from the sanatorium that she had a raised temperature every night, was suffering from night sweats and was losing flesh, and that laryngeal symptoms were present, he would suggest leaving the larynx alone. If success was to be obtained in the local treatment of tuberculous laryngitis, the cases must be selected with special reference to their general condition. If the temperature could be reduced to near the normal, the body weight maintained, and the general spirits of the patient were good, the laryngeal condition could be attacked with the greatest prospect of success.

Papillomata of Larynx from a Boy aged 6.

By HERBERT TILLEY, F.R.C.S.

THIS was the boy shown at the last meeting.¹ Intratracheal and extratracheal (tracheotomy wound) masses had since been removed and were now shown.

DISCUSSION.

The PRESIDENT said he once had a case at the hospital, a child, from whose larynx he removed a papilloma, yet in whom dyspnoea persisted. He therefore did tracheotomy, and then found a large papillomatous mass in the trachea, the removal of which was followed by the restoration of normal breathing.

Mr. TILLEY replied that the tracheotomy-tube was now out, the papillomata showed no signs of recurrence, and the child was now breathing through the larynx. If it had not been for the intratracheal mass he could have removed all the papillomata *per vias naturales* by the direct method.

A Vulsellum-catch Forceps for Firmly Securing Submerged Tonsils in the Operation for their More Efficient Removal.

By HERBERT TILLEY, F.R.C.S.

DISCUSSION.

Mr. TILLEY said that the objection which had been raised against the forceps was that one was apt to get out pieces of tonsil, and that the capsule was not entered. He had brought some specimens of tonsils removed that day in which a large portion of the capsule could be seen.

Dr. JOBSON HORNE exhibited a pair of forceps which he had had by him for very many years, but which he had seldom used, for a similar purpose. He claimed no originality in the instrument, which was an old Army bullet-extraction forceps. He could not claim originality, the instrument was registered; but he could claim gratification upon reading a paper on "Some Points in the Anatomy and Surgery of the Tonsils" (contributed to the Section of Laryngology of the British Medical Association at Sheffield in 1908 by a colleague in New Zealand, so that there had been no collusion) to find that Dr. James Hardie Neil used a pair of forceps, bearing the same registered

¹ *Proceedings*, p. 97.

number, to retract the pillar of the fauces in removing tonsils.¹ He (Dr. Horne) referred to this instrument partly to illustrate how, in the absence of a special instrument, an obsolete surgical instrument could be turned to some account, and partly to caution those not constantly removing tonsils against exercising traction upon a tonsil with a view of its removal by guillotine or bistoury. Cases dealt with in that way were liable to cause troublesome bleeding and were best dealt with differently.

Dr. DONELAN said that several years ago an American colleague operated on a case of enlarged tonsils in New Orleans, and in using a vulsellum drew a large artery into the guillotine and cut it. The bleeding was so profuse that the common carotid had to be tied in the neck before it could be arrested. It had been suggested that the internal carotid itself had been injured. Dr. Donelan thought some cases had been recorded in which the internal carotid had been wounded by a tonsil guillotine owing to too energetic pressure from outside.

Dr. FITZGERALD POWELL said, when these rare cases of severe hæmorrhage after tonsillotomy were quoted, one was apt to lose sight of the conditions which determined the bleeding, such as the constitutional state of the patient and alterations in the size of the blood-vessels, as in the case of an abnormally large artery to the tonsil. In these circumstances the bleeding was just as likely to be brought about whether a bistoury, a guillotine, or any other instrument was used.

Mr. CLAYTON FOX agreed that the teeth of the forceps shown were too small, and that the growth was liable to give way when traction was made. It was not unattended with danger, and traction towards the middle line rendered the vessels liable to be divided. He had not found it necessary even to steady the tonsils. He had always freed the tonsils from the anterior pillar, which was the obstacle to the tonsil obtaining a free position. The instrument was likely to be successful only in the hands of skilful men.

Mr. STUART-LOW said that he did not see the necessity for the instrument; he considered that it was a return to an obsolete method of operating. For the younger class of patients no other instrument than the guillotine was required in skilful hands, and in adults, unless the tonsil was deeply seated, no method could be better than enucleation.

Dr. SCANES SPICER said he did not think the grip part of the forceps was large enough. For twenty years he had used something like the instrument that Dr. Jobson Horne had shown. With his bigger instrument he pulled the tonsil out, and it was important to do so before cutting or snaring.

Dr. W. HILL thought Mr. Tilley's instrument could be improved upon at the grip end; he had had some forceps made on the principle of the eagle's claw for pulling out the tonsils, and he thought it would be found that they did not tear through the tissue like a small-toothed instrument. The other arrangement of the instrument was excellent, and was a great improvement on the scissors-like handle usually employed.

¹ *Brit. Med. Journ.*, 1908, ii, pp. 892-94.

Dr. DAN MCKENZIE agreed as to the probable usefulness of the handle, and he also approved of the close-toothed end. At the same time he had found that when the teeth were close together the tonsillar tissue resisted the closure of the forceps, and, besides, when the tonsil was freed from its attachments it sometimes slipped from the forceps and the grip was lost. If, on the other hand, the forceps were like an eagle's claw, with widely separated teeth, the teeth tore through the friable tonsillar tissue. Whatever forceps were used it was necessary to avoid dragging too much on the tonsil, because with any vulsellum one could, by pulling too hard, tear the tonsillar tissue. He did not think there had been an authoritative pronouncement in this country with regard to the position of the operation of enucleation of the tonsil, although abroad, particularly in America, it had come into popular vogue and had been the subject of several interesting discussions. There was no doubt that enucleation was, to a certain extent, employed in this country, but it had not so far received much prominence in the *Proceedings* of the Section.

Dr. PETERS asked whether the tonsils were dissected out with the assistance of the vulsellum, or so dissected out and then removed with the guillotine. He asked how the forceps compared with the blunt conchotome.

The PRESIDENT said that one of the greatest advantages was that with this instrument one could pass the ring of the guillotine or snare over the tonsil more easily than if it had a handle.

Mr. HERBERT TILLEY, in reply, said the chief objections which had been raised against the instrument were made by those who had not used it. In the case of tonsillectomy the question was where the blood usually came from in post-operative hæmorrhage. It came from low down on the posterior pillar of the fauces. If that were cut with the guillotine, or with a blunted hernia knife or a scalpel, there would be bleeding. Therefore the first essential for anyone who was operating on the tonsil was that he should have some fairly accurate knowledge of where the vessels of the tonsil were situated. If one fixed the tonsil with the vulsellum and drew it forwards and upwards, one saw the posterior pillar and could detach it from the gland substance. Then there would be no severe hæmorrhage. He could assure members that the forceps he showed them would grip the tonsil quite well, and not let it slip. It was because he had wanted forceps for this purpose that he conceived the idea of having several small claws, which would grip tighter than two larger claws. He had tried the latter type, but they were not successful. With regard to the question of the security of grip, all he could say was that he would invite anyone to use the forceps for a case of tonsillectomy which they might be contemplating. If they would accept the loan of his forceps he was quite sure that their doubts as to the capacity of the instrument for gripping and holding the tonsil would be dispelled, and the tonsil could be removed quite cleanly. The particular advantage of these forceps was that one had not to let the tonsil go when it had been loosened in the capsule, as was necessary when using the vulsellum with scissor handles.

**A Portion of Mutton-bone Removed by the Direct Method
from the Right Bronchus of a Lady in whom it had been
Lodged for Ten Days.**

By HERBERT TILLEY, F.R.C.S.

THE general symptoms of the patient were constant irritating cough, numerous râles over the right base of lung, and temperature 102° F. The removal was effected under general anæsthesia.

Tuberculosis of Larynx in a Girl aged 12.

By G. C. CATHCART, M.D.

THIS case was brought to the hospital last week complaining of sore throat and numerous glands in the neck. Four years ago the child had had a diphtheritic throat, but the mother said the doctor had told her that it was "the wrong bacillus." On laryngological examination it was impossible to see the larynx on account of the intense tumefaction of the epiglottis.

Opinions were invited as to treatment in so young a case.

DISCUSSION.

Dr. JOBSON HORNE said it was a remarkable case. He could not offer an opinion without further information about the condition of the lungs and a report upon an examination of the sputum. He hoped the case would be exhibited again at an early date.

Dr. FITZGERALD POWELL suggested that Dr. Cathcart might be asked to show the case again when he had got it under observation and had more information to give as to the sputum, &c.

Dr. SCANES SPICER said he hoped every member noticed the breathing in the patient. She breathed by distending the abdomen; there was no costal breathing at all.

Mr. CLAYTON FOX said that at first sight the case seemed to be tuberculous; the clubbed epiglottis was very typical. But it would be interesting to know the condition of the lungs. He would put the child in a sanatorium for six months, keep her under careful watch, and see her again; if then she was suffering from pain, he would operate on the epiglottis.

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Mr. HORSFORD said that no reference had been made to the condition of the pharynx; there had been much thickening of the posterior wall there, and, though the child was young, the appearance was very suggestive of hereditary syphilis.

Dr. DONELAN admitted the possibility of syphilis, which could be settled by a course of treatment. With regard to the sanatorium treatment of tuberculosis, he wished to call attention to the fact that in some sanatoria there was no regular examination of the larynx at all. He recently saw a laryngeal case which had been going from bad to worse for six months, in a well-known sanatorium, and the larynx had not been examined during that time. He thought the Section might well make some recommendation on the importance of the examination of the larynx at regular intervals in all cases of tuberculosis of the lungs.

Dr. W. HILL said he had seen perichondritis of the larynx in children which was at first suggestive of tuberculosis, but had proved to be nothing of the kind; in such cases it was sometimes found that there had been laryngeal trouble in connexion with measles, or some other specific fever, which had left a thickening.

The PRESIDENT said he thought it looked like tuberculosis of a mitigated type. Cases were seen in which it was difficult to draw the line between lupus and tuberculosis. He would be sorry to have to be pinned down to a definite diagnosis in the case. He suggested trying the von Pirquet test with tuberculin.

Dr. CATHCART, in reply, said he saw the case for the first time only on Wednesday week, and since then there had been a considerable change; there was no solution of continuity of the posterior wall when she was first seen. He examined the lungs, and found nothing wrong in them. The house-surgeon omitted to examine the sputum. He would have the von Pirquet test made the next day.

Case of Extreme Deflection of Triangular Cartilage, with Crest and Adhesions, and Anterior Prickle-shaped Spur, treated by Septal Fissure.

By L. H. PEGLER, M.D.

YOUNG man, aged 19, complained of recurring attacks of deafness, inability to breathe through left side of nose, constant cold catching, and a disagreeable sniffing which annoyed both himself and his friends.

Examination showed very marked deflection of the septum with ascending crest to left, extending from a thorn-like spine in front to

beyond the junction with the ethmoid plate posteriorly. The spur and anterior part of the crest were joined by adhesions to the inferior turbinal, the atrophied and indented condition of which still remains. A deep groove in right side of septum marks the angle of deflection. The right inferior turbinal was hypertrophied, but breathing was carried on fairly comfortably on that side.

Treatment consisted in sawing away the spur and crest under chloroform, followed by septal fissure with the author's fissure forceps, and, after right partial inferior turbinotomy, inserting a thick india-rubber splint. The septum was forced over by the finger after the incisions had been made, until the two cavities corresponded in capacity as they do now. The splint was worn quite comfortably for one week. The deafness, sniffing, and a troublesome watery discharge from the right free nostril have ceased, and the breath-way is perfect.

DISCUSSION.

Dr. COUBRO POTTER thought this operation differed from Moure's operation, as Dr. Pegler carried his incisions much further back; the upper one to the ethmoid perpendicular plate if necessary, and the lower well into the vomer. Moure's operation was confined to the quadrilateral cartilage alone; that was the essential difference between the two.

Mr. CLAYTON FOX preferred to adhere to the name "Moure's operation," and that was the procedure in this case. Moure never laid down that a man should limit himself to division of the quadrilateral cartilage, and that the perpendicular plate of the ethmoid or the vomer should not be encroached upon; one could use his own discretion in that matter. The present operation only differed from Moure's in that the spur was dealt with simultaneously with the main deflection, whereas Moure preferred to deal with spurs, &c., twelve days beforehand.

Dr. SCANES SPICER said the result was very good; the patient had a good nose, and there were no abnormal contacts. It was quite suitable for the operation. When the septum was bent at a sharp angle high up and in a narrow nose, and made contact there, it could not be touched by Moure's operation; the scissors could not be got there. But by submucous resection ample room could be gained to remove pressure in asthma cases.

Mr. STUART-LOW said that if a successful submucous resection had been performed there would not have been such a depression in the septum as existed on the right side. There was a groove where secretions could lodge and crusts form, and this was a distinct drawback compared with the perfect result usually obtained after submucous resection.

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The PRESIDENT said that, however fond members might be of submucous resection, it was well to have it brought before their minds that there were other methods of treatment. He did not know why the present operation was not called Moure's, as its principle was due entirely to him. The result on the side operated upon was very good indeed.

Dr. PEGLER, in reply, said he usually spoke of this procedure as "Moure's operation," though he had considerably modified it and extended its utility. His own incisions were different from those in the drawings doing duty for Moure's incisions one saw copied from book to book, not excluding the very latest, in which the cuts were represented in the cartilage alone, nearly parallel to each other, and not far enough apart. With the newest pattern of septotome, which he held in his hand, he was able to get below the crest of the superior maxilla and as far back as he required. The blades, though longer, were nearly parallel when apart so as to enter the nostrils without injury to the alar walls. One or two of the members had compared these cutting forceps to Asch's, but one blade of that surgeon's straight forceps had a blunt edge. The speaker had quite abandoned Moure's scissors, as well as his metallic splint, which was painful and unsatisfactory. As Mr. Fox had pointed out, he found it a great advantage to remove all crests, spurs, and thickenings at the main operation. With regard to the term "septal fissure," in reply to Dr. Horne, he first introduced it in his paper at the Leicester meeting,¹ as a convenient one to employ in order both to distinguish it from the submucous operation and indicate in some degree the nature of the method. In reply to Dr. Scanes Spicer, there could be no acute angle in the septum higher up than the ascending line of junction between the triangular cartilage and the vomer, but rounded deflections in the middle turbinal region were common, and these could be considerably modified by making the incisions ample enough. This case had been brought forward to remind the Section that, valuable as the submucous operation was—which he often selected himself for suitable cases—one could arrive at excellent results by older devices. As regards perforations, there was generally so much thickening on the site of spurs and crests that a good operator seldom need make one. The submucous operation would have been difficult and tedious here, a drawback which, he had always maintained, tended to a shirking of that operation; and when, as in this instance, the patient came to one principally for relief from recurring attacks of deafness, there was a great risk, in neglecting radical treatment, of the symptoms becoming confirmed. He thought he could show equally good results in posterior vomerine deflections. In reply to Mr. Stuart-Low, there could be no crusting in the groove affecting the intact mucous membrane, and if the latter were not quite obliterated it mattered nothing; after the submucous operation the groove was rarely quite lost sight of. For any temporary crust formation along the line of the lower incision a spray of the ungt. hydrarg. nitrat., diluted with almond oil and parolein, recommended by Lack, was of great service.

¹ *Brit. Med. Journ.*, 1905, ii, p. 1202.

**Case of Malformation of the Soft Palate and Uvula in a
Girl aged 16.**

By J. DUNDAS GRANT, M.D.

THERE is a perforation above and to the right side of the base of the uvula, but the nature of it is quite undecided. There is no such cicatricial tissue as would accompany a specific perforation, but a history of such traumatism as could have accounted for it—namely, an operation on the tonsils in early childhood.

DR. DONELAN said he had seen a similar case in which the tonsil was removed by a bistoury and vulsellum. The palate was wounded and afterwards sloughed. The same accident was apt to happen where forceps were unskillfully used for the removal of adenoids.

DR. JOBSON HORNE referred to a similar case which had come under his care, and which he had shown at the last meeting. In that case the uvula and soft palate had become adherent to the pharynx. He had since then operated for the relief of this, and the result, he thought, would be satisfactory. He would bring forward the case again at the next meeting.

**Case of Removal of the Hypertrophied Anterior Lip of the
Hiatus Semilunaris for long-standing Catarrh.**

By J. DUNDAS GRANT, M.D.

MISS H., aged 22, first came to see me in October, 1909, complaining of discharge from the nose of a muco-purulent nature of 8 years' duration; on account of this she was operated on for nasopharyngeal adenoids. The discharge, however, kept gradually getting worse, and for the last year she suffered from frontal headache, worse on the right side. There was tenderness on pressure at the upper and inner angle of the right orbit, and it was elicited that the headache became most marked soon after midday. There was hypertrophy of both middle turbinals, which were freely bathed in muco-pus, and there was a small polypus in the left middle meatus. On transillumination the various cavities were clear, except the right frontal sinus. The case was

apparently one of muco-purulent catarrh of the ethmoidal cells, and possibly of the right frontal sinus. I at once removed the anterior part of the right middle turbinated body, and in a week's time the discharge in the right nostril had almost entirely disappeared. Attention was then turned towards the left nostril, and a small polypus in the middle meatus and one in the anterior part of the middle turbinated body were removed. The favourable result obtained on the right side was not repeated, and it was only in January of this year, when I thoroughly cut away the hypertrophied anterior lip of the hiatus semilunaris, that real improvement took place; within a week after this was done the discharge was reduced almost to nothing.

DISCUSSION.

Mr. HERBERT TILLEY asked whether Dr. Grant removed the anterior part of the lip which was swollen. Also did he drain the antrum more thoroughly than the anterior ethmoidal cells? He was convinced that many of the cases of so-called nasal catarrh had no existence as such, but were due to chronic catarrh of the sinuses, particularly the antrum, and if the antrum were efficiently drained the catarrh would rapidly cease.

Dr. GRANT, in reply, said the hypertrophied lip was interfering with the outflow from the anterior ethmoidal cells, and possibly from the frontal sinus. The patient had an intense running from the nose, very slightly tinged with pus. He would call it essentially a catarrh of the ethmoid cells. He considered that the ventilation of the antrum would be increased considerably by the removal of the hypertrophy, though the effect on the drainage could only be slight. He recommended that it should be done purposively, as it might be the key to many difficulties.

Large Chronic Swelling in the Right Tonsillar and Epitonsillar Region, with an Enlarged Gland in the Neck. ? Sarcoma.

By WILLIAM HILL, M.D.

THE patient, a middle-aged woman, was seen to have a large swelling, the size of a tangerine origin, in the position of a quinsy. There was, however, no sign of acute inflammation, no pain or evident fluctuation on digital examination, and no difficulty in opening the mouth quite widely. There was an enlarged gland in the neck near the angle of the jaw of the size of a small walnut. This faucial swelling and the gland

had been present, according to the patient's statement, for about twelve months. This history and the appearances were suggestive of a slow-growing lympho-sarcoma of the tonsil. On questioning the patient, however, it was ascertained that the size of the tumour varied, and that it had enlarged and burst, discharging matter several times within the last twelve months. In order to determine whether a chronic abscess or a cystic condition might be present, a puncture was made into the palatal swelling with an aspirating syringe and needle, but the tumour appeared to be solid, and, after puncturing in several directions, only blood was withdrawn. The tonsillar fossa or crypta magna (the so-called supra-tonsillar fossa) was then forcibly probed to a depth of 1 in. in an upward and outward direction towards the palatal recess, and no abscess or cystic cavity was opened. On further digital examination the tumour seemed to be solid. Although the history was suggestive of chronic recurrent quinsy, the result of exploratory puncture and palpation pointed to the malignant rather than to the inflammatory nature of the swelling. A further examination would be made under an anæsthetic, and if the case proved to be malignant, Vohsen's operation (recently performed by Mr. Wilkinson in a similar case) might be undertaken.

DISCUSSION.

The PRESIDENT said it was a situation in which mixed tumours were often found which were susceptible of enucleation. It might be that the tonsil was projected into the fauces by the growth rather than infiltrated by it.

Mr. HERBERT TILLEY said he had seen a very similar case in which the cause was a buried calculus in the upper part of the tonsil. The patient had frequent attacks of quinsy, and had tried all sorts of treatment. In his last attack he got rid of a calculus the size of a green pea. He did not think the present case was a growth. He suggested that under a general anæsthetic the tonsil should be removed; even if the condition were lympho-sarcomatous, it would come away fairly easily, and in any case first relief could be secured without an extensive operation.

Dr. DONELAN suggested that, if it was not an embedded calculus, it might be a tumour of the carotid body. He had a case of glandular enlargement and inward pressure and ulceration of tonsil without involvement of skin, which was described by Mr. Hutchinson as a "potato" tumour of the carotid body. It ran a very slow course, but, though the external enlargement was very great, the skin never ulcerated. The patient died, and it was found to be an endothelioma originating in the carotid body. He had shown the growth here, and he believed a preparation from it was now in the collection.

Dr. HILL thanked members for their suggestions. He thought that as the swelling was so hard it might be malignant, although the history rather pointed to its being inflammatory; but he intended to examine thoroughly under an anæsthetic, and try to shell out the tonsil to clear up the diagnosis.

Dr. KREBBER (Baden) called attention to a tonsil instrument which had been devised by von Brünings consisting of a snare with a thick wire, and a lancet to fix the tonsil. The wire permitted of bending, and there was practically no loss of blood.

Dr. DONELAN showed:—

- (1) A curious septal deflection in a girl aged 11.
- (2) A large collection of mucous polypi removed from the nose of a man aged 50.

Laryngological Section.

May 6, 1910.

Dr. J. DUNDAS GRANT, President of the Section, in the Chair.

A Penny removed from the Gullet of a Young Girl.

By HERBERT TILLEY, F.R.C.S.

PATIENT, aged 14, swallowed a penny and applied to hospital thirteen days later because of difficulty in swallowing solid food. The coin was easily detected when the patient's neck was "screened." Under chloroform narcosis, dorsal position, head slightly extended, a medium-size bronchoscope tube was passed and the coin detected just below the cricoid region of œsophagus. The mucous membrane around the penny was very congested, in parts slightly ulcerated. After much difficulty in getting an instrument which would firmly grip the coin, the latter was finally removed with a Killian's long toothed-forceps. The patient left the hospital four days later.

DISCUSSION.

Dr. SCANES SPICER said it would be a great help if the exact measurements in centimetres of distance from the teeth were given in all such cases, so as to get conclusive knowledge as to why such bodies stick in the œsophagus in various regions. It was remarkable that such bodies as a penny should pass the sphincter and yet get lodged in a wider part of the gullet just below. It was, no doubt, the prominence of the first dorsal vertebra behind, the manubrial notch in front, and the first ribs externally, which, forming the superior orifice of the thorax, provided a sufficiently narrow channel at the best of times when packed with the trachea, gullet, thyroid gland, great vessels, nerves, and lymphatic glands. If the thoracic cage were depressed much compression and further obstruction of the œsophageal channel might ensue, and thus a constriction in the gullet be produced from outside in the superior aperture of the

thorax. This situation was also the lower limit of excursion of the cricoid cartilage, and he was of opinion that the chronic and recurrent exaggerated mechanical strains which were experienced at this isthmus in belly breathing were the cause of much of the chronic inflammatory and hyperplastic disease, and subsequently malignant degeneration, so common about the inferior border of the posterior cricoid arch, and in the area of excursion of the cricoid cartilage.

The PRESIDENT (Dr. Dundas Grant) asked whether the ulceration was so extensive that Mr. Tilley thought the old coin-catcher would have been a dangerous instrument.

Dr. EDWARD DAVIS said he had a case at Charing Cross Hospital, that of a child, aged 9 months, who swallowed a farthing. The diagnosis, by means of X-ray screen, was "a farthing in the left bronchus." A farthing would not go into the glottis, or at least through it. According to the history, that farthing had been in the œsophagus at the bifurcation of the trachea a month. He passed the œsophagoscope and could not see any ulceration, though the œsophagus was a little white in the position occupied by the coin. The farthing was easily extracted by gripping it with Patterson's forceps and removing it with the œsophagoscope.

Mr. HERBERT TILLEY, in reply, said there was no question about the ulceration; it was the first thing which became evident. On examination of the upper end of the gullet the mucous membrane was swollen and ulcerated, so that only a small segment of the circumference of the penny was visible. It was fixed rather tightly in the œsophagus, so that at first one felt in despair of being able to get it away, because on trying to grip it with Patterson's long forceps the jaw-joint of this instrument snapped. He therefore put the coin-catcher down, but felt afraid to slip it over the penny, lest he would not be able to pull it out. There was much difficulty with the oozing of mucus and the bleeding. That was checked by wool mops moistened with adrenalin and cocaine. The bubbles of mucus could be removed by applying a wad of wool dipped in ether.

Left Abductor Paralysis in a Man aged 64. Case for Diagnosis.

By H. J. DAVIS, M.B.

THE patient appears quite well. He lost his voice suddenly one month ago; the cords meet at the vocal processes only; there is no laryngitis. X-ray photograph of chest negative. The left pulse appears to the exhibitor to be slightly retarded (?).

DISCUSSION.

The PRESIDENT said he did not suppose there would be any difference of opinion as to the fact of the paralysis of the vocal cord. It was always difficult to decide whether a cord was in a position of adduction or in the cadaveric position. There was no tracheal tugging. Perhaps Dr. Davis would report the case later.

Mr. BARWELL said the left cord was in the cadaveric position, and that that was the reason the two cords did not meet, although the right cord came beyond the middle line. It was useless for members to guess what was the cause of the paralysis.

A Case of Excision of the Entire Tongue, with its Results.

By H. J. DAVIS, M.B.

THE patient, a man aged 45, who came a long way to London for the purpose, was anxious for the opinion of the Section as to the possibility of an artificial tongue in his case.

He came to the hospital in May, 1909, complaining of difficulty in speaking and slight earache on the right side. There was a small fungating, but not ulcerating, growth at the base of the tongue and some laryngeal catarrh. Under iodide and mercurial treatment the growth shrank. A piece was punched out three times, and on each occasion it was reported as not cancerous; nevertheless, it proved to be so, and in August, 1909, my colleague (Mr. Donald Armour) divided the jaw in the mid-line and removed the entire tongue. The movements of that organ were never impaired; it was nowhere adherent; and no glands were detected, but the growth invaded the base of the tongue from one side to the other as can be seen in the specimen—a very unusual one.

The patient's present condition is as follows:—

(A) He is well but thin. He speaks volubly, but is not easy to follow, and among several peculiarities of speech, all "f's" are pronounced "th"—e.g., he describes his age as "thorty-thive," &c.

(B) "He cannot bite properly"; "he loses his food as he never knows where it is in his mouth"; but food or liquids "never go down the wrong way," and this though the epiglottis is visible for $\frac{3}{4}$ in. standing vertically upwards. It will be noticed how insensitive the

anterior surface of the epiglottis is compared with the posterior or laryngeal surface, and also how large a buccal cavity appears without a tongue.

The exhibitor would be glad of opinions as to whether a rubber tongue or plate would improve the patient's speech and make his meals more comfortable.

DISCUSSION.

The PRESIDENT said the case upset one's ideas of the formation of the consonants; and it was worthy of more detailed consideration than could be given at the meeting. It was to him quite unexpected for a patient without a tongue to be able to utter the hard *th* as well as he did. His great difficulty seemed to be in those consonants which were stopped at back of the throat—*k* and *g*.

Mr. DONALD ARMOUR said he was much indebted to the Section for allowing him to discuss the case. It was referred to him by his colleague (Dr. Davis) at the West London Hospital, where the man had been attending the throat department. The striking thing about the tongue when he saw it was the great size of the growth, which occupied the dorsum and lateral surfaces of the tongue, being more extensive on the left side than the right. There was complete mobility of the organ in spite of the enormous size of the growth, and there was no glandular enlargement. At first he refused, from the clinical appearances, to accept it as epitheliomatous; and it was only after Dr. Davis had had three specimens examined that it was agreed the patient should undergo operation. The previous sections had been taken for examination from the surface of the growth. Before proceeding to the major operation he put the man under an anæsthetic and cut a wedge-shaped piece from the tongue at the growing border of the tumour. All the previous incisions which had been made in the growth had promptly healed; there was no ulceration following them. After the last incision he drew the edges of the wound together by means of a stitch or two, and healing occurred by first intention. Sections from the piece removed were submitted to several well-known London pathologists, and they at once declared it to be epithelioma. Therefore he proceeded to remove the entire tongue, which was rendered necessary by the extent of the growth. The operation was done by a median incision, through the lower lip, splitting the lower jaw, and retracting either half of it, and removing the tongue in the ordinary way, catching the lingual artery on each side as it was exposed. The hyoid bone was left perfectly bare to insure leaving no growth behind. Even during the operation no sign of glandular enlargement could be found, and none had been discovered since.

Mr. CHICHELE NOURSE said that one advantage which would accrue from a rubber pad placed in the floor of the mouth was that it would bring the food into contact with the palate during the process of eating. In that way the

patient would know by tactile sensations where the food was, and would probably be able to guide it towards the teeth and the buccal pouches. As it was, the patient complained that the food fell into the pocket formed by the floor of the mouth, and that he could not get it up to the teeth to be masticated.

Mr. ARTHUR EVANS said he had been dissatisfied with his attempts to get an obturator; he did not think a solid rubber foundation for the mouth would be good; it would not compare with the mucous surface in the mouth. He congratulated the surgeon on the excellent operation and the patient on the result, and advised them to "leave well alone."

Dr. DAN MCKENZIE said if there was not too much cicatricial contraction in the floor of the mouth it might be advisable to try the effect of paraffin injection into the floor.

The PRESIDENT again suggested that the phonetic aspect of the case should be specially studied. He was surprised to find the patient unable to sound the letter *f*, which was a labial, and that he substituted for that the lingual-dental *th*. It became a question whether there was any neurotic disturbance. Perhaps Dr. Davis would like one or two other members to combine with him in studying the case.

Dr. DAVIS replied that the man's inconvenience seemed to be not very great. The only food he could take with ease was minced food. He was unable to feel where the food was, and sometimes he swallowed it before he was prepared. Although the epiglottis was quite unprotected, no food ever went down into the larynx. It was usually supposed that, in swallowing, the bolus received an impetus from the base of the tongue. The man spoke with great volubility. In the days of the Inquisition, when it was the intention to punish and silence a man for preaching against the faith, the executioner removed as much of the tongue as he could and as far back as he could reach; but after a time the victims talked as well as ever, a fact which was attributed to a special act of Providence. That he should pronounce *f* as *th* was contrary to what one would expect, and he agreed with the President that these factors in the case were of great interest.

Case for Diagnosis.

By H. J. DAVIS, M.B.

THE patient is a man aged 25, with a large vascular tumour implicating the left tonsil and lateral pharyngeal wall. It seems of the nature of a venous (?) angioma. There is a large vascular swelling on the neck on that side, evidently in communication with it. If the patient lowers his head the tumours at once distend.

DISCUSSION.

Mr. ATWOOD THORNE asked whether the condition had got worse lately, or improved. What operation, *if* any, did Dr. Davis propose to do?

The PRESIDENT thought Dr. Atwood Thorne's *if* was a very important factor. Radium had been used successfully for small vascular growths.

Dr. DAVIS, in reply, said he only saw the case for the first time recently, when it was sent to him for an opinion. He supposed it was a large venous angioma. It did not pulsate. He did not think tying the carotid would very much relieve the patient. The man said he was at the London Throat Hospital twelve years ago and had a tonsil removed, when bleeding was so furious that they detained him in the hospital. He now had no inconvenience, except that when he lay down at night the growth in the mouth swelled up. The condition was best left alone, though if hæmorrhage occurred from any cause it would be fatal.

**Fungating Mass on the Epiglottis, with Implication of the
Larynx, in a Man aged 45.**

By H. J. DAVIS, M.B.

TRACHEOTOMY was performed nine weeks ago, and the tube is still being worn. Three weeks ago the patient was readmitted for severe arterial hæmorrhage from the growth. He is up and about at home again, attending to his duties and eating and smoking, and he appears to have very little inconvenience.

Dr. DAVIS added that the bleeding was arterial and only arrested with great difficulty. He now refused any further operation, and he felt that he could not advise otherwise.

Epithelioma of the Larynx.

By E. A. PETERS, M.D.

G. T., AGED 74, plumber, complains that he has lost his voice for nine months. He considers he has had something the matter with the throat for two years. There is a warty growth involving the right false and true cords. The right cord does not move and œdema has attacked the

corresponding arytaenoid. Direct œsophagoscopy shows the posterior surface of the cricoid to be free. The lungs are very emphysematous.

The question is whether operation is advisable.

DISCUSSION.

The PRESIDENT said that he was not in favour of operation.

Mr. ARTHUR EVANS said it was such an extensive growth, the glands were so much involved and the man so aged, that he would leave the case alone, especially as the patient breathed and swallowed fairly well. He did not think the man could stand such a large operation as excision of the whole malignant mass, and no other operation would be of any use. When the time came that the patient began to have difficulty in taking fluid nourishment, gastrostomy should be performed.

Dr. PETERS, in reply, thanked members for their opinions, which supported his own inclination to do nothing in the case.

Model of the Upper and Lower Jaws and Impression of the Roof of the Mouth from a Young Man aged 19.

By A. R. TWEEDIE, F.R.C.S.

THE alveolar arch on the right side has a less pronounced outward curve than that on the left, and the hard palate on the right side is narrower, and also at least $\frac{1}{4}$ in. higher than that on the opposite side. The patient has a deviation of the nasal septum to the left, and the apex of the nose is tilted to the right.

The specimen is shown to elicit an expression of opinion as to whether the asymmetry of the upper jaw and palate has any causal relation to the septal deflection. The post-nasal space is perfectly healthy and contains no adenoids or other obstruction. The patient has never had any operation on "the nose or throat" and is not a buccal-breather.

DISCUSSION.

Mr. BARWELL asked whether Mr. Tweedie could give any explanation why the palate should be wider on the less obstructed side. In the last specimen shown by Dr. D. R. Paterson,¹ he believed of congenital occlusion

¹ *Vide* p. 114.

of the choana, the palate was higher on the side obstructed; whereas in this case it was higher on the opposite side. He asked whether Mr. Tweedie had had casts made of people who had comparatively normal noses, and whether there was not some asymmetry of the palate in many apparently normal persons.

Mr. TWEEDIE replied that the patient had no facial paralysis, and that the specimen was shown at the Oxford meeting of the British Dental Association last year, when it was suggested that the flattening of the right alveolar arch was probably due to the premature extraction of the temporary teeth. He (Mr. Tweedie) submitted that this flattening of the outward alveolar curve had necessitated the palate finding accommodation for itself by an upward excursion, and thus tilting the maxillary crest over to the left. He had seen only one other case like it, but, unfortunately, had not had a cast prepared.

Party-wall Pharyngeal Cancer.

By WILLIAM HILL, M.D.

FEMALE, with a malignant tumour involving part of the pharyngeal aspect of the laryngo-pharyngeal party-wall. The parts involved are the posterior surface of the cricoid plate, more especially to the left, the anterior part of the adjacent pyriform fossa, and the pharyngeal surface of the left arytaenoid and left aryepiglottic fold. The œsophagus has been found by endoscopic examination to be free from disease. The case seems eminently suitable for Gluck's type of operation of complete laryngectomy *plus* partial excision of the deep pharynx.

DISCUSSION.

Mr. ARTHUR EVANS said that last year he had a case much more advanced, so that the whole upper aperture of the œsophagus was involved with the cricoid surface. He urged the patient to go home and have nothing done, and when the time came that there was difficulty in swallowing to have a gastrostomy done. But she came back and urged him to attempt to remove it. He accordingly first performed a gastrostomy, and later removed the whole of the larynx, a part of the pharynx, the cervical œsophagus, and as much of the thoracic œsophagus as his fingers could reach. Into a fistulous opening which he had made immediately below the hyoid bone he inserted one end of a flanged empyema tube, and the other end was connected by rubber tubing with the gastrostomy tube. This was seven months ago; there was no sign of recurrence and the patient took all her food—liquid and solid—by

mouth, and no onlooker would guess that an india-rubber extra-thoracic œsophagus took the place of the normal one. He was so encouraged by this result that he urged Dr. Hill to operate at once in this case, as here there was every possibility of being able to save sufficient pharynx and œsophagus to enable the patient to take food in the normal way.

Mr. WESTMACOTT said gastrostomy should not be deferred to the time when the patient could not swallow liquids or foods; it should be done early, when the patient could eat and drink comfortably. The want of success of the operation was largely due to the asthenia set up by the patient being half-starved. He asked whether it would not do to remove the posterior wall of the larynx, and leave the thyroid cartilages alone.

Dr. H. J. DAVIS said he did not think the larynx was involved to such an extent as to necessitate its entire removal. She would be better off if nothing but gastrostomy was performed.

The PRESIDENT said the reason for removing the larynx, in his opinion, was that the pharynx could not safely be removed without it. The safe course was to remove both at the same time, and bring the trachea to the surface.

Epithelioma of the Larynx in a Man aged 69, fourteen months after Operation.

By CHICHELE NOURSE, F.R.C.S.Ed.

PREVIOUS to operation the patient had been troubled with hoarseness and partial loss of voice for five months. At the time of examination the voice was hoarse, deep-toned, and weak. The whole larynx was inflamed, and there was a sausage-shaped red growth occupying the anterior two-thirds of the left vocal cord. Thyrotomy was performed on February 18, 1909. The left vocal cord, the left ventricular band, and the left arytenoid were removed. The thyroid cartilage was completely ossified, and could not be sutured at the close of the operation. It was brought together by suturing the perichondrium. Recovery from the operation was rapid; the patient has remained well ever since, and has gained flesh.

Microscopical examination of a specimen removed before the operation and of the tumour after removal proved that it was an epithelioma. A gland the size of a split pea removed from the external surface of the crico-thyroid membrane showed no signs of malignant infection.

Two Cases of Radical Frontal Sinus Operation, to show the Conditions Nine and Sixteen Months after Operation.

By STCLAIR THOMSON, M.D.

It is frequently suggested that the only satisfactory way of judging of the results of treatment of sinus suppuration is to show the patient some time after all treatment has been discontinued. The two following cases illustrate the satisfactory results which may be arrived at.

Case I (Mrs. M.).—This patient had the radical Killian operation carried out on the left frontal sinus on November 19, 1908. The case was exhibited before the Section on December 4, 1908 (vide *Proceedings*, ii, p. 51). The patient's left maxillary antrum was also operated on, and a suppurating ethmoidal region cleared away. The patient has not been up for inspection for more than a year; during that time she has remained quite free from the headaches (for which she begged to have the operation performed) and never requires a nose lotion.

Case II (Mr. D. E. J.).—This gentleman gave a history of eighteen months' nasal suppuration. He had received endo-nasal treatment, and had tried the effect of a visit to the Cape. The left frontal sinus contained very foul pus. On August 5, 1909, a radical Killian operation was carried out on the left frontal sinus and an endo-nasal operation on the left maxillary sinus. The patient was out for a walk on the thirteenth day, and returned to the country within three weeks. The depth and height of the sinus can be judged from the skiagram and from the depression on the forehead; yet there is no marked disfigurement, and the patient now enjoys excellent health and perfect freedom from all his symptoms.

DISCUSSION.

Dr. WATSON WILLIAMS said he could not help being struck with the excellent result in the male patient. Evidently it was an enormous sinus, illustrating what doubtless had been brought home to all operators: that if the anterior wall of such a large sinus was removed nothing could prevent some supra-orbital depression, whatever operation was chosen. Apart from that, there was practically nothing to be seen in the way of scar or other defect, and Dr. StClair Thomson must be congratulated on the result obtained.

The PRESIDENT asked whether Dr. Thomson had considered the question of filling up the depression in any way, as, for instance, by the introduction of paraffin.

Dr. STCLAIR THOMSON replied that he had submitted the idea of paraffin injection to the patient, who said he did not think he would bother about it; he was a married man! The æsthetic point turned up very often in their debates, but the much more important point was that these patients were absolutely free from suppuration. He thought the success was largely due to clearing out the ethmoid thoroughly.

Ulceration of the Epiglottis, probably Epitheliomatous.

By HAROLD BARWELL, F.R.C.S.

THE patient, a man aged 63, has had increasing discomfort for five months: tickling cough, occasional pain shooting to left ear, and slight dysphagia. The epiglottis is red and thickened, and an ulcer is visible on its laryngeal aspect. The infiltration appears to involve the base of the tongue on the left side.

DISCUSSION.

The PRESIDENT said the disease had extended to the lateral wall of the pharynx, and the pain was as much due to the disease of the pharyngeal wall as to that of the epiglottis. He did not think removal of the epiglottis alone was likely to give a satisfactory result.

Mr. BARWELL replied that he was sure the growth was beyond the epiglottis; the only point was as to whether it was within the reach of operation. The relatives were anxious to have something done, if at all possible.

Case of Extrinsic Malignant Growth of Larynx.

By P. WATSON WILLIAMS, M.D.

THE patient was shown at the January meeting of the Section.¹ There had been a considerable increase in the glandular enlargement in the larynx, but only a slight increase in the amount of the growth.

¹ *Vide* p. 60.

146 Pegler: *Traumatic Perforation through Hard Palate*

There was no ulceration. Opinions were divided as to whether it was an epithelioma or an endothelioma. The exhibitor had not felt justified in removing a portion for diagnostic purposes.

Traumatic (post-operative) Perforation through the Hard Palate, communicating with the Floor of Left Nasal Fossa and Maxillary Antrum.

By L. HEMINGTON PEGLER, M.D.

THE patient was a woman aged 36. Suggestions as to feasibility of a plastic operation were invited.

DISCUSSION.

Dr. BRONNER said there was a history of discharge from the left nostril for some time. The operation performed had probably been for empyema of the maxillary antrum. As the surgeon who operated would probably be interested in the case, he should suggest that the patient should be sent back to him.

Mr. BARWELL said he did not think a plastic operation would be of much advantage, for while the patient wore her dental plate she got no food into the nose. He counselled leaving it alone.

Infiltration of the Right Half of the Larynx of Obscure Nature, in a Woman aged 34.

By J. DUNDAS GRANT, M.D.

THERE is an irregular swelling of the right half of the epiglottis and of the right ary-epiglottic fold; it seems to be firm in texture, and the surface appears papillated and of a reddish tint; it is of such extent as completely to conceal the right vocal cord. The rest of the epiglottis is slightly enlarged, and there is in the left glosso-epiglottic space a smooth, rounded, sessile swelling, apparently growing from the lingual surface of the base of the epiglottis. The patient complains of hoarseness of three weeks' duration, and has had slight huskiness and cough for six months; there are no physical signs in the chest. Her blood is being submitted to Wassermann's test, but apart from that there is no evidence of specific dyscrasia.

DISCUSSION.

DR. GRANT added that it might be lupus, but it did not agree with the picture of any disease of which he knew, so far. Possibly the Wassermann reaction would help the diagnosis, and a portion might be removed for microscopical examination. There was no pain. He hoped to give a more detailed account at the next meeting.

Dr. WATSON WILLIAMS said he had examined the case, and thought it might be an infiltrating malignant growth ; probably the removal of a portion would settle the diagnosis.

Odynphagia in a Tuberculous Subject, without any obvious Disease of the Larynx.

By J. DUNDAS GRANT, M.D.

THE patient, a woman aged 35, complains of loss of voice, and during swallowing of severe pain running up to the left ear. In the left hyoid fossa, on the outer surface of the ary-epiglottic fold, there is an extremely ill-defined sessile area of œdema, but otherwise the larynx is normal in appearance, and the movements of the vocal cords are in no way diminished. There are physical signs of tuberculosis in the chest, but for the moment any tuberculous condition giving rise to the odynphagia must be quite latent. Von Pirquet's cutaneous tuberculin test has been applied, but the result is still being awaited.

New Growth of Right Tonsil invading the Surrounding Tissues.

By ARTHUR EVANS, M.S.

A.S., MALE, aged 40, first seen on May 5, 1910, complaining of pain on swallowing. In the region of the right tonsil, and apparently originating in it, is a new growth which has invaded the soft palate up to the mid-line, the lateral pharyngeal wall posterior to the tonsil, the anterior pillar of the fauces, and the soft structures covering the inner surface of the angle of the lower jaw. From here the new growth

passes on to the adjacent portion of the tongue. Lymphatic glands on the right side of the neck are enlarged, and presumably secondarily infected. The symptom complained of—viz., pain on swallowing—was first noticed one month ago.

DISCUSSION.

Dr. H. J. DAVIS said he regarded the case as epithelioma, and considered it operable.

Mr. EVANS, in reply, said he proposed to tell the man what the operation consisted of, the risks which it entailed, and leave the decision to him. It meant taking away a part of the jaw, the tonsillar region, a large part of the palate, a good deal of the pharynx, a part of the tongue, and the glands on the right side of the neck. It was a large operation, but the age of the patient was only 40. If, after having it explained to him, he decided to have the operation done, he (Mr. Evans) would bring the patient up at a future date.

Laryngological Section.

June 3, 1910.

Dr. J. DUNDAS GRANT, President of the Section, in the Chair.

Case of Obstruction of Both Nostrils.

By ANDREW WYLIE, M.D.

GIRL, aged 11, said to have had the obstruction all her life. The tonsils and adenoids were removed twelve months ago; her mother states that no operation was ever performed upon the nose. Marks of hereditary specific disease are seen; the Hutchinson teeth are characteristic. The hearing is normal. The septum is thickened, and the anterior ends of both inferior turbinals are firmly adherent to it except at the lower border on the left side, where a fine probe can be passed. The obstruction is chiefly anterior, as posterior rhinoscopy shows a normal condition.

The exhibitor intends to remove both anterior ends of the turbinals, dissecting them from the septum, and for several weeks afterwards keeping the passages patent by means of large drainage tubes.

DISCUSSION.

The PRESIDENT (Dr. Dundas Grant) said much of the obstruction in the left nostril was due to the septum, which had got away from the middle line. The patient was apparently the subject of the syphilitic dyscrasia.

Dr. WESTMACOTT said he thought there had been ulceration of a congenital syphilitic nature, and a raw surface had been left, which had resulted in adhesion to the surrounding parts.

Dr. WYLIE, in reply, said that adenoids had been removed several years ago, but the mother told him no operation had ever been performed on the nose, and that since birth there had been an obstruction of both nostrils. He had seen the case for the first time three weeks ago, and had not prescribed anti-specific remedies.

Laryngeal Paralysis following Partial Removal of the Thyroid Gland.

By SOMERVILLE HASTINGS, F.R.C.S.

A WOMAN, aged 41, had suffered from goitre for fifteen years. The whole gland was extensively involved, but the left side more than the right. On May 2, 1910, the greater part of the tumour was removed piecemeal by resection-enucleation. In doing this the right superior thyroid artery was divided. Loss of voice and slight dyspnoea were noticed immediately after the operation, but owing to an attack of broncho-pneumonia the examination of the larynx was deferred to May 24. The patient is unable to cough; she speaks with much waste of air in a feeble, unmusical voice. There is slight expiratory stridor. The cords are pale; they are held immovable in incomplete adduction, and flap forward in expiration. The contraction of the crico-thyroid muscle can be felt during attempts at phonation.

DISCUSSION.

Mr. HERBERT TILLEY asked whether, during the operation, the surgeon saw either of the recurrent laryngeal nerves. It had been regarded an axiom that if those nerves were not seen during the operation, they would not be injured. A surgeon, who was also a skilled anatomist, recently asked him to see a case in which the same operation had been done on a child, and she also had bilateral recurrent paralysis. That operator said he did not see the recurrent nerves, and therefore he did not think he could have wounded them, because they lay deep. In the present case the nerves might have been cut or might simply be bound in the post-operative adhesions.

Dr. FITZGERALD POWELL said he thought that there had been a very considerable portion of the thyroid gland removed, probably all of it, and he thought it possible that myxoedema would develop before long and necessitate the administration of thyroid extract. He could not quite understand the

operation described as "piecemeal enucleation," and thought that such a method would be more likely to injure the recurrent nerves than if the half of the gland were removed in the usual way. Damage to the recurrent nerves arose by tearing or cutting, by inclusion of the nerve in the ligature of the inferior thyroid artery, or by its being pressed upon by the contraction of scar tissue in the healing of the wound. The latter might possibly be the reason of the paralysis in the present case. He had seen some cases of less severity than this one, yet the voice improved after a time, and he thought careful and sustained electrical treatment would do good.

Dr. WATSON WILLIAMS asked whether examination of the larynx was made before operation. Some of the cases of abductor paralysis of the vocal cord, due to pressure by an enlarged thyroid gland, were overlooked, because the symptoms were not such as were likely to attract the attention of the patient, and the dyspnoea was attributed to compression and narrowing of the trachea by the practitioner, hence the possibility of paralysis of the abductors of the vocal cords might not be suspected. Thus in some of the cases of vocal cord paralysis following operation, the paralysis had existed before operation. In every case of goitre which was going to be operated upon, the larynx ought to be examined before the patient was submitted to operation.

Dr. DAN MCKENZIE said he had heard that, in removing the thyroid gland, one should look for the recurrent laryngeal nerve so as to avoid it. Anatomically that nerve was subject to considerable variations. It sometimes passed in front of, and sometimes behind, the inferior thyroid artery, a fact which supported the view that, in order to avoid damaging it, the nerve should be identified, if possible, in operating on the thyroid.

Mr. HORSFORD said he regarded the paralysis as complete. The cord was in the cadaveric position rather than that of adduction. The movement was merely due to the blast of air in and out; therefore he regarded the prognosis as bad.

The PRESIDENT said there was very little evidence of the expiratory stridor which was present before, and which he thought was due to the typical scabbard-shaped narrowing of the trachea. He thought there was likely to be but little change in the laryngeal paralysis.

Mr. HASTINGS, in reply, said that he was not present at the operation, and was only given an opportunity for examining the larynx some three weeks later. The operator had told him that neither recurrent laryngeal nerve was seen. He believed that no examination of the larynx had been made before operation, but quite agreed that this was desirable in all cases. The respiratory stridor was certainly becoming less marked, but the condition of the larynx had not changed since he first saw it. He did not think that the cords moved at all. He quite agreed that there was apparently a slight adduction, especially of the left cord, when the patient said "ah." But if they watched carefully they

would notice that "ah" was, in all cases, said during inspiration, and the natural tendency of the indrawn blast of air impinging on slackened cords is to approximate them. He was greatly indebted to the members of the Section for their suggestions as to treatment. He would see that they were carried out.

**Tuberculosis of the Larynx, with Extreme Odynphagia,
relieved by Injection of Alcohol into the Left Superior
Laryngeal Nerve.**

By J. DUNDAS GRANT, M.D.

THE patient, a young woman, when seen on May 24, complained of such intense pain in swallowing during the preceding week as to prevent her from taking any nourishment except in the form of thin liquids. She had had signs of tuberculosis of the lungs for two years. When first seen by the exhibitor on April 5, 1910, she complained of hoarseness and some degree of sore throat. There was infiltration of the left ventricular band, on which was an irregular tuberculous outgrowth; both the vocal cords were infiltrated, and there was a serrated thickening in the inter-arytænoid space. She was ordered auto-inhalation of anæsthesin and orthoform by means of Leduc's tube, but this did not prevent the extreme pain complained of during the week preceding May 24. On that date the infiltration of the left ary-epiglottic fold was accompanied by superficial ulceration. One cubic centimetre of 80-per-cent. alcohol, containing a trace of eucaine, was injected into the region of the superior laryngeal nerve between the hyoid bone and the upper margin of the thyroid cartilage, by means of Schloesser's syringe. The needle was somewhat coarse in structure, and sharpened to a much more obtuse angle than in ordinary hypodermic syringes, so as to render it incapable of puncturing the superior laryngeal artery, and it had a mark to indicate the depth of $1\frac{1}{2}$ cm. On the night of the injection her swallowing was already easier, and when seen three days later she stated that she could swallow quite well and thought her voice was rather better. The exhibitor had obtained an equally satisfactory result in the case of a female patient in the wards of Brompton Hospital.

DISCUSSION.

Mr. MARK HOVELL drew attention to a simple means of relieving the swallowing difficulty. It consisted in placing the palm of the hand over each ear, with the fingers pointing upwards, and making very firm pressure. The greater the pressure the greater the relief of pain. As a rule mothers who knew the method seemed afraid to exert sufficient pressure. When he was a boy, aged 11, he had diphtheria badly. An emetic was administered, and he brought up a cast of the larynx, trachea, and both bronchi. Swallowing was so painful that he refused to take food, but the kitchen-maid, on hearing this, asked if she might hold his head, and adopted the measure which he now recommended, which enabled him to swallow without pain. This simple measure was effective in all cases of painful swallowing, whether the cause be malignant disease, scarlet fever, diphtheria, phthisis, tonsillitis, or any other affection.

Dr. WATSON WILLIAMS said he had found the procedure suggested by Mr. Hovell of great use in practice. It brought great relief in quinsies and tonsillitis, enabling the patient to swallow without pain. He usually instructed the patient to compress firmly with the thumb or forefinger immediately in front of the tragus. Probably this effect was due to compression of certain branches of the fifth nerve.

Dr. DONELAN said the syringe interested him because he brought out a syringe which was described and figured in the *Lancet* in 1897 as the first all-metal sterilizable syringe. It was the first application of the plunger-piston to surgical uses. He had refrained from patenting it on ethical grounds, but it had been not only imitated but patented in Germany, France, and other countries, without any acknowledgment. The present syringe was merely one of the imitations in all but the nozzle.

Mr. HOWARTH asked whether Dr. Grant had difficulty in finding the nerve, and how he knew when he was exactly over it.

Mr. CLAYTON FOX asked how long the action lasted, and if, in cases where the sphincter action of the larynx was much impaired, it would not be better to divide the superior laryngeal nerve.

Dr. GRANT, in reply, said the point about the syringe was the stumpy end; it was bevelled off at a more obtuse angle than usual in hypodermic syringes, so as to diminish the risk of puncturing the laryngeal artery. He had been much indebted for the information given by Mr. Hovell, and had used it constantly in acute inflammation, chiefly of the tonsils, but he did not think it applicable to prolonged conditions like tuberculosis. In answer to Dr. Howarth, one had first to decide on which side to inject. In this case it was the left side which was most painful. The patient was lying on the right side, and one could push up the larynx. The nerve was between the hyoid bone and the upper

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margin of the thyroid cartilage. He could produce pain by pressing on a particular spot, which no doubt corresponded to the site where the superior laryngeal nerve penetrated the thyro-hyoid membrane. He introduced the needle and felt about until it touched a spot which sent a pain shooting up to the ear. Then he injected gradually 80 per cent. of spirit. He had not been tempted to do the excision of the nerve mentioned by Mr. Clayton Fox, as it was much more serious than injection. The duration of relief varied, but sometimes it was for weeks or months. The present patient was injected only twelve days ago, but he had a patient in Brompton Hospital who was injected a month ago, and the relief still lasted. That was borne out by the results in the treatment of trigeminal neuralgia.

Case of Probable Late Secondary Specific Pharyngitis, with
Nerve Symptoms.

By J. DUNDAS GRANT, M.D.

THE patient, a woman aged 24, complained of difficulty in getting her words out and occasional loss of voice, which had been most marked within the last two or three months. She had had some soreness of the throat for the last nine months. In the pharynx were found symmetrical vertical red splashes on the pillars of the fauces, at the upper edge of which were slightly-marked opalescent elevations, especially on the right side. There was slight enlargement of the posterior cervical glands, which she stated to have been previously more considerable. There was ptosis of the right eyelid, with occasional diplopia on looking up. Liquids sometimes regurgitated through the nose. The palate was paretic. The *Spirochæta pallida* was not found in a scraping from the throat, but Wassermann's test was positive, confirming the opinion that the condition was a late secondary specific involvement of the levator branch of the right third nerve, also of the motor nerves to the palate. There was no history suggestive of diphtheria to be elicited, and the knee-jerks were active.

DISCUSSION.

Dr. FITZGERALD POWELL asked the President whether this was in his opinion a "peripheral neuritis," and not a central nervous lesion. A case of great interest had come under his care lately in which a girl, aged 22 to 24,

who had had a specific history, showed symptoms of labio-glosso-pharyngeal paralysis—acute bulbar paralysis. She died suddenly in a fit two days after being seen. The palate, lips, tongue and pharynx were paralysed.¹

Mr. A. L. WHITEHEAD thought it was more likely to be peripheral neuritis. Probably Dr. Powell's case was one of basal meningitis.

Dr. DONELAN said there might be deeper glands causing pressure. He had seen an exactly similar type of face in connexion with pressure from a goitre on the cervical sympathetic, with ptosis.

Dr. GRANT, in reply, said he did not think any brain centre would produce the combination of symptoms. Such a paralysis of the palate was more usual in a toxic lesion like diphtheria, where there was local neuritis. Wassermann's test was positive. He had seen ptosis characteristic of cervical sympathetic mischief, but it was not so extreme as in this case and was accompanied by a drawing-up of the eyebrows and increased sweating. He believed this was an isolated involvement of the branch of the third nerve, coincident with some lesion of the pharyngeal branches of the vagus supplying the soft palate.

Hypertrophy of the Lingual Tonsil, with Impairment of Singing Voice, improved by a Snaring Operation.

By J. DUNDAS GRANT, M.D.

THE patient, a young woman, complained of tickling cough, which occurred soon after beginning to sing. The larynx presented a slight degree of catarrh and imperfect apposition of the vocal cords (paresis of the internal tensors). There was very marked hypertrophy of the right half of the lingual tonsil, overhanging and obviously touching the epiglottis. A portion of this was removed with the snare, and the patient, when seen four days later, stated that the cough had been very considerably diminished and her singing greatly facilitated. The exhibitor has notes of a case of a lady student of singing, in whom the removal of a portion of the hypertrophied lingual tonsil was followed by greatly increased facility in singing and the immediate addition of several notes to the upper part of her compass.

¹ A post-mortem examination was made by Dr. Spilsbury, pathologist to St. Mary's Hospital, and no gross lesion or any disease was found to account for the condition or for death.

DISCUSSION.

Dr. SCANES SPICER said he had had several cases of hypertrophied lingual tonsil, and it was almost impossible to snare them, except with the galvano-caustic snare. He had not succeeded with the cold snare. The guillotine of Brady was most successful where the epiglottis was caught.

Mr. HORSFORD asked if there was any harm in cauterizing the lingual tonsil.

Dr. GRANT replied that he had not been so successful with the guillotine as with the cold snare. It was difficult to engage the lingual tonsil in the guillotine, and he had a snare with a special curve for the purpose. It was not necessary to remove the whole, but only the portion pressing on the epiglottis. In answer to Mr. Horsford, he said Sir Felix Semon reported an experience of the occurrence of phlegmonous inflammation after cauterizing that region, though he (Dr. Grant) believed the case to have been most exceptional.

Epithelioma of the Right Vocal Cord in a Man aged 60, seven months after Operation; no Recurrence; Development of Cicatricial Substitute for Vocal Cord.

By J. DUNDAS GRANT, M.D.

THE patient was shown in November, 1909.¹ There was subsequent development of a reddish granulation tumour at the anterior commissure. This was completely removed by the galvano-cautery and the subsequent application of chloride of zinc, being simply granulation and not new growth.

Tuberculous Ulceration of Epiglottis apparently healed by frequent use of Electro-cautery.

By JAMES DONELAN, M.B.

THE patient, aged 44 (shown on May 7, 1909),² has had pulmonary disease since 1904. Open-air treatment in various places ever since. Pulmonary condition quiescent, no pyrexia. He was seen first by

¹ See p. 17.

² *Proceedings*, 1909, ii, p. 141.

exhibitor in January, 1908. The epiglottis was a mass of tuberculous ulceration and infiltration; apices of both arytaenoids slightly infiltrated. Treatment at first by means of lactic acid, guaiacol, absolute silence, &c.; much improved. Shown in May, 1909, with a view to taking more energetic measures. Punching-out ulcerated part, amputation, and curetting were suggested, also electro-cautery. In view of extension of disease below attachment of epiglottis, and from experience of other cases, the galvano-cautery was selected. Patient had fifteen or sixteen cauterizations with various-sized burners. The disease had apparently disappeared from the epiglottis and arytaenoids in November, 1909, when he contracted influenza and bronchitis. There was very profuse expectoration from the old pulmonary foci, and the epiglottis again showed some small ulcers on the left side of the old cicatrix and its lower margin. The electro-cautery was resumed in January, 1910, and applied on ten or twelve occasions. There are now no evidences of active disease in the epiglottis. The general appearance of the larynx has undergone no change during the past twelve months. The small, central pin-head elevation has similarly remained unchanged.

A Series of Specimens and a Case illustrating Diseases of the Ventricle of the Larynx.

By W. JOBSON HORNE, M.D.

SOME twelve years ago and subsequently I have reported cases and pathological investigations drawing attention to the ventricle of the larynx as a site of infection (tuberculosis, diphtheria) which might be overlooked, and I have also shown specimens illustrating innocent and malignant tumours of the ventricle. In the earlier part of the session, in the discussion of a case exhibited as possibly one of prolapse of the lining membrane of the ventricle, I ventured to remark that that condition was in my experience an extremely rare one, and that the appearances simulating it were due to a neoplasm. Owing to the distension of the ventricular band, the matting of the parts together, and the obliteration of the landmarks, it is not always easy, from the image in the mirror, to decide at a glance whether in the living subject the appearances presented are to be attributed to a prolapse of the

mucous membrane lining the ventricle, or to a tumour, cystic or solid, presenting itself at the mouth of the ventricle. The appearances presented are more readily studied in macroscopic and microscopic sections, and therefore I venture to exhibit again specimens and preparations which were exhibited many years ago. The following specimens are shown:—

(1) A series of microscopic sections cut vertically through the entire length of one side of the soft parts of the larynx, illustrating the development of an innocent neoplasm, dependent from the roof and tending to present itself at the mouth of the ventricle. This specimen was accidentally met with whilst investigating a series of larynges obtained in the Pathological Institute of Berlin by Professor Virchow, and given to me with his approval by Professor Kanthack, for the purpose of further investigating pachydermia laryngis.

(2) A section of the right half of the larynx, showing true prolapse of the mucous membrane lining the ventricle. The detachment of the membrane has been brought about by ulceration and destruction of the cartilage secondary to a gumma. This specimen illustrates that prolapse of the ventricle, although speaking generally is more a "tradition," is at times actually an entity.

Case.—The patient, a woman aged 48, has suffered from hoarseness for many years; in fact, according to her own statement, since childhood. She is a street seller of fruit and flowers, and attributes her hoarseness, and probably quite correctly, to that trade. The larynx presents swelling and distension of the right ventricular band, and when first examined by myself the appearances suggested a projecting body from the right ventricle obscuring a view of the vocal cord in its entire length. Both cords moved equally. I shall be glad to learn the impression created in the minds of others by an examination of the larynx. The patient is the subject of other general medical ailments, doubtless having a bearing upon the matter, into which space will not allow me to enter.

DISCUSSION.

Dr. SCANES SPICER, discussing Dr. Horne's series, said he had lately been working at diseases of the ventricle area in connexion with the genesis of cancer, and the present cases afforded a good opportunity of testing the views he had advanced. Dr. Horne's first specimen, with a diagram, was described in the *Proceedings of the Laryngological Society*, 1898, v, pp. 96-99, showing how an innocent growth arose from the roof of the ventricle. That specimen,

as far as he had studied it, did not show the complete relations of the site of origin to the cartilaginous parts or if the latter were ossified, and so did not assist in the elucidation of the question he was raising. But in the same paper Dr. Horne said he had often found, in subjects dead of tuberculosis, tubercle bacilli in the ventricle in cases which showed no macroscopic disease of the larynx, and that "in the posterior and inferior parts of the ventricular bands" he had found tuberculous disease commencing. That was in the area of play of the arytenoid, and round and external to the vocal process. That agreed with what he would expect, in accordance with the views he had put forward, that the hard parts, where they worked in or against the soft tissues with excessive force in abnormal actions of the body, produced in those soft parts a state of lessened resistance and undue irritation which favoured invasion by tubercle bacillus and also malignant degeneration. He claimed that the fact that tuberculous invasion was proved by Dr. Horne sometimes to commence at the site indicated, suggested that that was a point of lessened resistance, due to excessive mechanical irritation, as was to be expected on the theory the speaker had advanced. With reference to Dr. Horne's second specimen, he would only remark that bare cartilage was exposed in, and some had been exfoliated from, the cavity of the broken-down gumma. Although the exciting cause of the gumma was the parasite of lues, was it not probable that the movements of the cartilaginous edge (was it ossified?) were the determining cause of the gumma appearing at that spot? In the patient shown there was "something" in the situation of the ventricle. He (Dr. Spicer) believed he could detect this "something" moving about between the ventricular band and the vocal cord. Whether it was eversion of the ventricle or a neoplastic growth, it was impossible to say. He would invite members to notice whether the points of commencement of neoplastic degeneration were, or were not, in the immediate area of play of one of the hard spots of the endoskeleton. During the last year Dr. Bashford had stated again that cancer only occurred among the vertebrata. The speaker pointed out that it was this class alone which had an *endoskeleton*, a *portal system*, and a *lymphatic system*. It was the abnormal friction of soft tissues on some hard part of the endoskeleton, especially in abdominal breathing, which, in his belief, caused the localization of cancerous degeneration. When such abnormal friction was very great there was more likelihood of inflammatory or infective conditions, and, when mild, there were chronic congestions, and, if persistent for years, neoplastic conditions. It was not improbable that, as this patient got older, and if a toxæmia arose from the abdominal pool, the condition would become clinically malignant, although now, structurally, it might have the same histological characters as Dr. Horne's case, previously published.

The PRESIDENT said it would be interesting to hear opinions as to the laryngoscopic appearances where the growth was situated. He believed it came from above the vocal cord, but he could not see the vocal cord at all, though he tried, by expiratory phonation, to bring it into view. If the vocal

cord was present, expiratory phonation usually brought it into view by causing recession of the ventricular bands. Possibly it grew from the interior of the ventricle. He believed the ventricle was a very favourite seat for tubercle bacilli, and often in cases of catarrh of the larynx, where inspissated mucus came between the vocal cords, interfering with the voice, the mucus was secreted in the ventricles, and there was probably catarrhal disease inside the ventricles more often than could be clinically decided.

Dr. HORNE, in reply, said it was difficult for him to follow Dr. Spicer's hypothesis. He did not regard the vocal process itself as a common site of origin of tuberculosis in the larynx. When the vocal process became involved, it did so by extension; it was the most resistant spot in the larynx. But the ventricle lent itself most suitably to the development of tuberculosis because the sputum shot up into the larynx lodged in, or ultimately found its way into the ventricle. There was thus a typical culture tube, and the condition spread to surrounding parts. The vocal process was immune from tuberculosis unless attacked by extension, because the spot was free from glands. He thought the case was one of a growth springing from the ventricle itself, and that it was attributable to straining the voice.

Tumour of Post-nasal Space in a Man aged 27.

By H. FITZGERALD POWELL, M.D.

THIS man was first seen on May 25, 1910. He complained of nasal obstruction and some difficulty in swallowing. He stated that he had influenza five weeks ago, which was accompanied by sore throat. Fourteen days ago he noticed a swelling on the right side of his palate and right tonsil, which was painless, but caused him difficulty in swallowing and obstructed his breathing through the right nostril. The influenza and sore throat cleared up in about ten days or a fortnight, but the swelling remained and was increasing. May 31: The patient has been under treatment for a week, 20 gr. of pot. iod. being given three times daily. He says he thinks the swelling and nasal obstruction is decreasing, though no specific history can be obtained. On his admission on May 25 an exploratory puncture was made into the swelling, but no pus was evacuated. A portion of the growth was removed for microscopical examination. Dr. Briscoe examined the portion of the growth removed, and I am much indebted to him for the slides. The report on the microscopic section was that it resembled

in appearance the condition found in sarcomata. June 2: I examined the man again, and, though he himself thought that it was smaller, I could not detect much difference in the growth, which filled the right side of the post-nasal space, was hard and somewhat elastic, and was pushing the palate before it.

DISCUSSION.

The PRESIDENT asked what data were obtained by palpation. Could Dr. Powell make out that the growth was behind the palate, and not inside it? Also, from what part of the growth was the specimen taken? There seemed to be some breaking-down in the growth, but the great rapidity, as stated, appeared to be incompatible with the development of a tumour, and more suggestive of an inflammatory or specific lesion.

Mr. CLAYTON FOX said he thought the swelling was in the palate. There seemed to be no sign of growth springing from the nasopharynx. He thought it was inflammatory, but it was difficult to say whether pus was present.

Dr. WATSON WILLIAMS asked whether there was any syphilitic history. The right lateral wall of the nasopharynx seemed distinctly infiltrated by this nasopharyngeal growth, but it seemed to be impossible, by mere inspection or palpation, to say how far the soft palate was infiltrated or simply pushed forward. He thought it was probably a sarcoma.

Dr. PEGLER said that the section under the microscope showed a mass of lymphoid tissue, and that the growth was a lympho-sarcoma.

Dr. FITZGERALD POWELL, in reply, said the patient came under observation on May 25, complaining that five weeks previously he had influenza with sore throat. This cleared up, but a fortnight later he felt a lump in his throat, causing difficulty in swallowing and obstructing the right nostril. A swelling bulging forward the soft palate, principally on the right side, was observed, and with the finger a hard elastic mass was felt behind the palate and filling up the post-nasal space. An exploratory puncture through the palate into the swelling revealed no pus. The growth appeared to be growing from the base of skull and lateral wall of post-nasal space. While exploring with the finger a portion of the mass was broken off, about the size of a small pigeon's egg, which was examined by Dr. Briscoe, the pathologist, who reported that it had the appearance usually found in sarcoma. The growth was felt to be growing from the naso-pharynx, and was not in the palate; the finger could be passed round between the growth and the palate. On examination with the mirror the growth obstructed the view of the posterior orifices of the nose and post-nasal space. There was no specific history, but he was put on 20 gr. of pot. iod. three times daily. The man thought the growth smaller, but he (Dr. Powell) saw no appreciable difference in it. He intended watching the case for a few days and would give due attention to the opinions expressed.

Case of Bilateral Abductor Paralysis from Central Nerve Disease.

By DAN MCKENZIE, M.D.

MALE, aged about 56. The larynx presents the picture of bilateral abductor paralysis, with incipient paralysis of the internal tensors. The cords are approximated when at rest, but they do not come fully into contact during attempts at phonation. He has frequently suffered from dyspnœa during the last two years, and has run the gauntlet of several attacks of acute glottic spasms; but, although fully aware of the risk he was running, he has steadfastly refused to submit to tracheotomy. He belongs to a family of hæmophiliacs. There is also at times some difficulty in swallowing liquids; the mobility of the tongue is impaired, and articulation is affected to some extent. The pupils are unequal and respond little, or not at all, to light. The knee-jerks are active and there is no Rombergism. The symptoms therefore point to a lesion in the bulb. For some time, also, he has been annoyed by a lack of control over his emotions; he laughs or weeps for quite trifling reasons. There is no luetic history. The Wassermann reaction is negative. He has been taking pot. iod., and more recently mercury.

Mr. CLAYTON FOX said there seemed to be some paresis of the orbicularis oris and of the tongue, but there was not so much paralysis of the latter, nor was there the degree of difficulty in swallowing usually associated with involvement of the nucleus ambiguus. He thought the case might be one of incipient bulbar paralysis.

Loss of Voice with Dyspnœa in a Woman aged 26.

By W. STUART-LOW, F.R.C.S.

THE patient was sent to the clinic in April last for loss of voice, which had been gradually developing for six months, and dyspnœa (especially on exertion) also increasing. The face shows a condition which has reached its present proportions in three years, having commenced as a small raised spot on the side of the nose. The pharynx and

palate exhibit scars, and the entrance to the larynx is reduced to the size of a pencil. The epiglottis is scar-bound laterally and anteriorly, the arytenoid apices being dragged forward and the ventricular bands shortened and approximated. There is no history of syphilis nor phthisis, and Wassermann's test, as reported upon by Dr. Wyatt Wingrave, does not support the theory of specific infection.

Even with rest in bed the inspiratory dyspnoea became so severe three weeks ago that a low tracheotomy was performed. She has greatly improved since, some oedema of the larynx having disappeared, and her general health is much better. She is now being treated with Donovan's solution.

DISCUSSION.

The PRESIDENT regarded it as a severe case of lupus.

Mr. STUART-LOW, in reply, said that although the pathological evidence was not clear that this unusual condition was the result of a blending of two dyscrasie—viz., lupus and syphilis—yet the therapeutical test bore this out, since the case had improved when anti-syphilitic remedies were given. The administration of iodide of potassium had to be temporarily discontinued, however, because oedema of the narrow space to which the laryngeal entrance was reduced supervened, giving rise to severe dyspnoea. Since a low tracheotomy had been performed the patient had been perfectly comfortable, the anti-syphilitic measures having been given with safety and the local conditions, both on the face and in the larynx, having greatly improved. He (Mr. Stuart-Low) wished to emphasize the great value of early tracheotomy in such cases.

Congenital Absence of the Bony Part of the Palate in a Girl aged 16.

By L. A. LAWRENCE, F.R.C.S.

THERE is no gap in the mucous membrane. The uvula is slightly bifid. The case looks rather like a cured cleft palate, but no operation has ever been performed on her.

The PRESIDENT said it was interesting in connexion with the important article in that month's *Journal of Laryngology*,¹ showing how often there was a defect in the bony palate without necessarily loss of mucous membrane.

¹ Kelly, A. Brown, "Congenital Insufficiency of the Palate," *Journ. Laryngol.*, 1910, xxv, pp. 281-300.

Case of Ulceration of Palate.

By L. A. LAWRENCE, F.R.C.S.

PATIENT, female, aged 44, widow. Superficial ulceration in patches on hard palate and gum on left side; ulceration of upper lip, right side; small outgrowth in intra-arytænoid space of larynx; weak voice. No history of specific disease. Improvement under treatment with iodide of potassium.

Tuberculosis of the Larynx, with considerable Dysphagia, treated and relieved by Congestion Hyperæmia.

By WALTER HOWARTH, F.R.C.S.

THE patient first came under notice complaining of pain on taking food. The epiglottis and arytænoids were so swollen that it was not possible to see the interior of the larynx. For the last three weeks the patient has been wearing, for twenty-two hours out of the twenty-four, a fairly tight elastic band round the root of the neck. This band is fitted with a pad, which lies in the hollow between the cricoid and the top of the sternum; the band can be tightened by a buckle at the back of the neck. Under this treatment the swelling has so far diminished that an easy view of the larynx is obtainable; and, further, the patient has much less difficulty in swallowing his food than was formerly the case. The patient is having no other treatment, and is pursuing his daily work as a brass finisher. The exhibitor has several other cases under treatment, but they are at present combining it with vocal rest at sanatoria.

DISCUSSION.

The PRESIDENT said he had tried it without producing the beneficial effect he anticipated, but his friend, Professor Juracz, recommended it strongly and he (Dr. Grant) would certainly give it a further trial.

Mr. HOWARTH replied that in one case he had tried the method of injecting alcohol with little result, and, though he had followed Hoffmann's technique, supposed that he must not have reached the nerve. More recently he had been trying the present method of congestion, and in several cases it produced a marked effect on the dysphagia. In this case the treatment could only be palliative, but in earlier cases it might be curative. He believed that good results were being obtained in Dr. Fraenkel's clinic in Berlin.

Large Globular Dilatation of the Upper Third of the Œsophagus above a Malignant Stricture.

By WILLIAM HILL, M.D.

MALE, aged 47. Difficulty in swallowing solids, unless very well masticated, commenced about two years ago. One year ago neck observed to swell up for half a minute after drinking a glass of water, and portions of an ordinary meal were sometimes regurgitated "on stooping forward." Has lost about 1 st. in weight during the last year, and troubles in swallowing have increased, though he can still manage solids well masticated. The swelling in the neck after food has gradually got larger. In October last year Dr. Hargreaves took the patient to Dr. Rowden, of Leeds, who found, by a meal of bismuth porridge and an X-ray photograph (fig. 1), that the globular, pouch-like dilatation of the gullet extended down to the suprasternal notch and measured $5\frac{1}{2}$ cm. in each diameter. A recent skiagram by Dr. Orton (fig. 2) shows that the dilatation of the gullet is now somewhat pear-shaped, measuring 9 cm. in its vertical diameter and $6\frac{1}{2}$ cm. transversely. On June 1 an œsophagoscopic examination was made, and the remains of the bismuth meal taken two days before were removed by repeated lavage and suction. The continuation of the œsophagus was found as a tight, œdematous, ulcerated stricture, commencing 26 cm. from the teeth. This was dilated by graduated bougies, and a soft rubber œsophageal catheter (6 mm. diameter), fitted with a silver style to prevent it being coughed up, was inserted. The tight part of the stricture was 3 cm. in length. The patient is being fed through this catheter, which will be retained for a week to bring about further dilatation of the stricture,

and an attempt will then be made to insert a permanent Symond's funnel. The œsophagoscopic examination clearly showed that the case was not one of dilatation above an aneurysm, nor of a large pharyngeal diverticulum (the so-called œsophageal pouch), as had been suggested by



FIG. 1.

Dilatation of œsophagus. Skiagram taken October 1, 1909.

others. A dilatation of this size in the cervical and upper thoracic œsophagus following a malignant stricture is quite exceptional, if not unique.



FIG. 2.

Dilatation of œsophagus. Skiagram taken May 30, 1910.

Dr. SCANES SPICER said it was generally accepted that one did not have dilatation of the œsophagus as the direct result of an antecedent cancerous stricture. One of the commonest sites for cancerous degeneration to take place, however, was the contracted lip of a stricture of inflammatory origin of the whole lumen, or the marginal lip of one of the sacculated diverticula of the œsophagus.

Endolaryngeal Carcinoma.

By WILLIAM HILL, M.D.

MALE, aged 57, with an ulcerated cancerous growth involving only the left ventricular band and left vocal cord, with slight *enlargement of the glands in the neck* near the left side of the thyroid cartilage. The case was too advanced for thyro-fissure, though very suitable for radical removal. Opinions were invited as to whether hemi-laryngectomy would be considered sufficient in this case. The exhibitor favoured total laryngectomy.

PROCEEDINGS
OF THE
ROYAL SOCIETY OF MEDICINE

VOLUME THE THIRD

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1909-10

MEDICAL SECTION



LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1910

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MEDICAL SECTION.

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Medical Section.

October 26, 1909.

Dr. J. MITCHELL BRUCE, President of the Section, in the Chair.

On Oxaluria and the Treatment of Calcium Oxalate Deposit from the Urine, with a Method for the Solution of Calcium Oxalate Calculus whilst in the Urinary Passages.

By ROBERT MAGUIRE, M.D.

It is proposed to discuss in this paper—

- (1) Certain matters concerning the nature and mechanism of oxaluria in general ;
- (2) A case which illustrates some of these and also oxalate stone formation, and in which it is claimed that an oxalate stone was dissolved in the urinary passages ; and
- (3) Some laboratory experiments in which the same process was successfully repeated *in vitro*.

PART I.

It is known that the fresh normal urine contains a certain amount of oxalic acid, which can be determined as such after destruction or alteration of its salts. Its amount is small and by no means easy of estimation, but has been fixed, most recently by Dunlop [5], at a medium of 0·0172 grm. for the twenty-four hours' secretion. Calcium, also, in comparatively loose combination, is present in the normal urine in quantity variously estimated at about 0·3 grm. to 0·4 grm. in the daily output. The affinity between this acid and this base is so keen that, if they exist together in solution, combination must be assumed to occur unless some powerful influence should intervene. Again, calcium oxalate is so extremely insoluble in water, or in a solution of ordinary salts so dilute

as is the urine, that the compound would be expected to show itself as a deposit in one of its well-known crystalline forms, octahedra or dumb-bells, immediately after the urine is passed. Yet the normal person shows no such regular deposit of calcium oxalate from his urine, although from time to time those who have no other apparent departure from health excrete urine which throws down a crystalline shower of this salt. Such deposits may be nothing more than a portion of the physiological content of the urine, extruded under the influence of ill-understood or unknown alterations of physical suspension or chemical solution. Not infrequently such showers alternate with others of crystalline uric acid suggesting that each may have its cause in an alternating disorder of metabolism. Further, these small vagrant and alternating showers of calcium oxalate on the one hand, and of uric acid on the other, are at times accompanied by slight nervous and dyspeptic symptoms, scarcely severe enough to attract the attention of their subject, usually, like the deposits, of short duration, and having no apparent connexion with the general health. Yet they must be due to a disordered mechanism and, in higher degree, may be of more serious import. Obviously, it is important to know the relation, if any; between these symptoms and the crystalline showers, and also why the very insoluble calcium oxalate, small in amount though it be, should be held up in ordinary urine, and yet at times deposited. The conditions just described do not overpass the limits of healthy variations, but may easily do so. The normal small excretion of oxalic acid, in spite of certain views to the contrary, is probably an accident or incident in the metabolism of carbon towards a higher or a more oxidized form. If there should occur such changes in the body mechanism as would cause excessive production of calcium oxalate or of its immediate precedents, rather than of an otherwise developed and soluble carbon compound, then the normal means for maintaining calcium oxalate in solution in the urine would be overcome and deposit would be determined.

Calcium oxalate is practically insoluble in any solutions except those of the mineral acids, yet it may form no deposit in the presence of acid phosphate of sodium, to which the acid reaction of the urine is due. It is probably this salt which holds up from precipitation the small amount of calcium oxalate present in normal urine, and it continues to do so even if the acidity be neutralized. The exact method by which this is brought about is unknown, but I suggest that we have to do, not with an ordinary process of solution, but with the formation of a soluble double salt of the two bases. Dunlop thinks that

other substances in the urine may have a similar effect, but he was unable to find them.

Any precipitation of oxalate of calcium in the urine, beyond the slight occasional showers mentioned above, must be considered to be due to an exaggerated production, and is generally accompanied by an exaggeration of the nervous and dyspeptic symptoms. The deposit and the symptoms were grouped together by Begbie [2] into a "clinical entity" under the name of Oxaluria. Usually the symptoms have been thought to be the result of the conditions producing the deposit, but it is one object of this paper to show that the oxaluria is, sometimes at least, caused by the dyspepsia, and that this in turn is produced by mental overstrain, of which the nervous symptoms are an indication. In some cases, and, as I would maintain, in that to be presently described, mental and physical strain is the primary factor, causing atony of the stomach and colon, and a dyspepsia with obscure chemical results; the chemical changes which occur as the result of the dyspepsia then lead to metabolic disorders which result in the excessive production and secretion of calcium oxalate, and to the deposit of the salt from the urine.

The association of a dyspepsia with oxalate urinary deposit has many times been insisted upon, but never, as I think, satisfactorily explained. Dunlop asserts that it is simply an acid dyspepsia with excessive secretion of hydrochloric acid, which favours the absorption of oxalic acid from food-stuffs. This assertion appears from his paper to be based on nothing more than a resemblance between the symptoms of such acid dyspepsia and those met with in oxaluria—an unreliable groundwork for an opinion. Against it is the fact that the dyspepsia which accompanies oxaluria is usually of the atonic kind, and, as in the case to be described, the stomach may be relaxed and dilated. Such a condition is not associated with excess of hydrochloric acid, but rather with fermentation and a late dyspeptic formation of lactic, butyric, and other fatty acids by decomposition. Dunlop gives two experiments to show that also lactic acid favours the absorption of oxalic acid from food-stuffs, but the conditions of the experiments were very different from those which obtain when lactic acid is produced in a dilated stomach by decomposition and fermentation some time after a meal. On the other hand, atonic dyspepsia with its accompaniments, and also true acid dyspepsia, in by far the majority of cases, are unattended by a deposit of oxalate in the urine, no matter what the diet may be; while, again, oxaluria, and even calcium oxalate calculus, may at times occur without any symptom of

dyspepsia having been perceived. For this reason, I remark that the dyspepsia, when present, has obscure chemical results, upon which the copious literature of the subject gives absolutely no information. Yet in many cases, as in that to be discussed, a causal relation between the dyspepsia and the oxaluria can scarcely be doubted.

The primary source of the deposited calcium oxalate has been much discussed, and most of the numerous views upon the subject which have been put forward may be thus summarized:—

(1) Uric acid may by decomposition be transformed into oxalic acid, either within the system or in the urine after it has passed the kidneys.

(2) The oxalic acid may be produced from gelatin or mucus, either introduced with the food or formed in the stomach.

(3) The oxalic acid contained in a large number of food-stuffs may be simply absorbed and re-secreted.

(4) Deficient oxidation may cause retarded metabolism of any or all the food-stuffs, and so bring about the production of oxalic acid, instead of a more highly evolved compound.

All these views have been argued pro and con by many authors, usually with insufficient facts at their disposal, but the last view—that of retarded metabolism—first put forth by Beneke [3], has been the most generally accepted, and seems at least to be the most consistent with the clinical appearances. Of recent years, however, the third view—that the oxalate is derived from the oxalates of the food-stuffs—which was originated by Cantani [4], and supported in this country by Dunlop, has received renewed attention. Its principal basis seems to be that a larger number of vegetables than we suspected contain calcium oxalate, and that a purely meat diet (Cantani), a purely milk diet (Dunlop), or a diet of bread without crust (Esbach) [6], will cause the disappearance from the urine of calcium oxalate of which a deposit had previously been found. Obviously, the premises are at least uncertain, for it may equally and even better be argued that these diets have simply removed the dyspepsia whose peculiar chemical results have caused the formation of oxalic acid. Further, other observers, especially Auerbach [1], Wesley Mills [10], and Petteruti [11], have found even a considerable amount of oxalate deposit in the urine after a diet entirely devoid of oxalates. Toepfer [13] contributes an interesting, though not conclusive, observation. He ascertained that in one of the large hospitals of Vienna not more than 5 to 6 per cent. of the patients showed any oxalate deposit in the urine. On certain days the general dietary for all patients contained spinach, sorrel, or other vegetables in which is a considerable quantity of

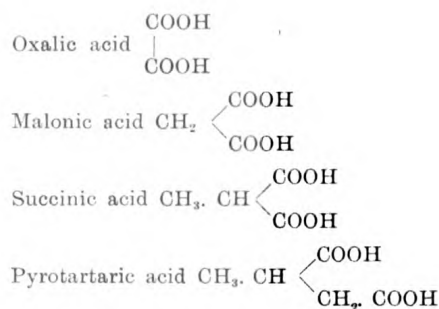
oxalic acid, yet on those days there was no increase of oxalate deposit amongst the patients.

Those who hold the "alimentary oxaluria" theory seem to assume, whenever the point is in any way mentioned at all, that the oxalate of calcium is absorbed from the stomach, passed as such into the blood, and carried as such to the kidneys, to be there simply excreted. But Owen Rees [12], whose view of the production of oxalate from already excreted uric acid was probably wrong, held strongly that it was impossible for such an insoluble salt to exist in the blood, and here he was certainly correct. There is a mechanism for maintaining a small amount of calcium oxalate in solution in the urine, but this does not exist in the blood, and if it did it would be powerless to maintain in solution for carrying purposes the larger amount found in the urine in oxaluria when the mechanism of the urine itself is overpowered. Insoluble calcium oxalate can no more pass through the walls of the stomach than through those of the kidney, and still less can it exist in the blood. The presence of oxalate of calcium in the stomach, on the one hand, no matter how introduced there, and in the urine on the other, even in relative quantities, is no argument for the supposition that there is a direct connexion by the blood between the two "loci" of the salt, without the intervention of an intermediary compound. Nor is it less improbable that the intermediary compound is oxalic acid itself or one of its soluble salts. There are only two facts which even apparently support such a view. Sir Alfred Garrod [7] found calcium oxalate in the serum of a blister, but his patient was already suffering from the uric-acid diathesis, Bright's disease and pleurisy. Sir Alfred Garrod himself did not conclude that the oxalic acid had existed in the blood, and there was no need for the elaborate discussion of Esbach to show that the occurrence could be otherwise explained. Cantani is said to have found oxalic acid in the blood, but, as I have not had access to his original writing, I cannot criticize the conditions of the observation.

Further, in spite of one or two opinions to the contrary, the amount of oxalic acid occurring at times as a calcium salt in the urine, if present in the blood in solution, and probably therefore in combination with another base, ought to call forth the serious nervous and other phenomena of oxalic-acid poisoning, while the nervous symptoms found in oxaluria are by no means of this nature. Dunlop, indeed, considers them to be identical with those of simple acid dyspepsia. Esbach himself swallowed 6 grm. of oxalic acid, and while it is certain that the whole of this was not absorbed, a considerable amount must have passed into the system,

6 Maguire: *Oxaluria and Calcium Oxalate Deposit*

for he found in his urine no less than 0.181 gm., the highest determination ever made by the Neubauer method. Yet he says that he experienced no inconvenience from his huge dose, which is surely impossible if so large a quantity of oxalic acid had circulated in the blood in soluble form. Again, a soluble salt of oxalic acid circulating in the blood must of necessity reach the tissues which contain an excess of calcium. Calcium oxalate would be formed and, for want of a solvent, would remain there permanently. But while this salt has been found in various secretions, it has never been detected in the tissues; therefore, if oxalic acid is really carried in the blood, it must be in a non-poisonous form, and, although we have no direct observations at our disposal concerning the nature of this form, some experiments of Heymans [8] have given very suggestive results. Oxalic acid is the simplest compound of its series, consisting merely of two carboxyl groups directly united. Heymans found that as the series is ascended by adding one or more methyl groups so as to form—



the poisonous properties of oxalic acid are gradually but greatly diminished, until the compound finally acts merely as an acid. Thus, on the frog, the poisonous dose of oxalic acid being represented by 1 cg., for malonic acid it is 2—2.5 cg., for succinic acid 4—5.5 cg., and for pyrotartaric acid 6—6.5 cg. Ascent in the scale of series is very common in metabolism, and may provisionally at least give us an explanation of the state in which oxalic acid is conveyed.

Whatever may be the form which oxalic acid assumes for the purposes of circulation, the presence of calcium oxalate in the urine is not a mere effect of filtration or diffusion of a pre-existent substance through an animal membrane. Certain observations by Kobert and Küssner [9] have a most important bearing upon this point. They found in experimental poisoning by oxalic acid that crystals of calcium oxalate were seen in the convoluted and straight tubes of the kidneys,

where actual secretion takes place, but none in the glomeruli which are concerned chiefly with filtration and diffusion.

It would from these arguments seem probable that the effect of the dyspepsia when present is not to promote absorption of oxalates, but to produce one or more substances which so alter the inner metabolism as to provide the kidneys with such material as will produce calcium oxalate.

However distressing to the patient the symptoms which accompany oxaluria may be, they are nevertheless not of a serious nature in themselves. But when once existent as a deposit in the urine, calcium oxalate is a dangerous body, since its crystals can accumulate to form a calculus, either with or without the intervention of a mucous or colloid base, as insisted upon by Ord. The stone may consist entirely of calcium oxalate with colouring matter, as, I think, in the patient whose case is to be described ; but more commonly the calculus contains a uric-acid nucleus, or may even be composed of alternate layers of oxalate and uric acid. The latter feature reminds one of the alternating showers of the two compounds already mentioned as occurring in simple deposit form, and this point, as will appear, is not without importance in regard to treatment. The shape of the oxalate stone is characteristic—"mulberry-shaped," as it is usually called, though "spiked" would be a more correct term. Though generally of small size, it is known to be the most irritating and painful of all calculi, is recognized as the hardest with which the surgeon can have to deal in lithotripsy, and has hitherto been considered out of the reach of all solvent methods, which, it must be admitted, have not always been so successful as was hoped, even with more promising materials.

I would maintain that in the following case (which illustrates some of the theoretical points mentioned above) I succeeded in dissolving an oxalate calculus whilst existent in the higher urinary passages of a patient, and with removal of all the consequent symptoms.

PART II.

Mr. X., aged 44, is an "insurance explorer," a profession which necessitates, at times, severe mental and physical fatigue. In the spring of 1907 he passed through a period of great overwork, anxiety, and sleeplessness, while occupied in settling the insurance claims arising from the West Indian earthquake, and in consequence suffered from what he called "nervous dyspepsia," a diagnosis which was probably more correct

than he supposed. On returning to England a year later, he placed himself under the care of a physician, who treated his dyspepsia, and later discovered a pronounced oxaluria, then, it is said, in the form of a dumb-bell deposit. Subsequent hæmaturia and severe pain in the right flank, extending to the right testicle, made it probable that a calculus had formed in the upper urinary passages of the right side, but several X-ray examinations failed to demonstrate this. After consultation with another physician the patient was advised to go to Vichy in the month of August, where during a period of three weeks he took alkaline waters and a diet mostly composed of vegetables. Towards the end of the period he passed a small stone, which proved to be composed of oxalate of calcium, but his local symptoms were in no way relieved, his dyspepsia persisted, and his general weakness, ill-health, and loss of weight had increased. He consulted me first on September 11, 1908, on his return to London.

The patient looked ill and worn. He complained of feeling weak, of loss of appetite, and of flatulent dyspepsia. The bowels were irregular, at times constipated, and at times loose. The pulse was of low tension and feeble force, the tongue was coated with a white fur, the stomach and the large intestine were distended with gas, and, about three hours after a meal, splashing on percussion was detected over the areas of the stomach and cæcum. The patient complained of constant aching pain in the right flank, extending to the region of the sacro-iliac synchondrosis and down the line of the ureter to the right testicle and the inner part of the right thigh. The slightest movement accentuated the pain, even the act of turning in bed; whilst to walk a hundred yards would cause a "stabbing" pain in the same parts, which made further progress impossible without rest. Micturition was frequent, but not painful. The urine passed per diem varied in quantity between 35 oz. and 45 oz. It was neutral or slightly alkaline in reaction, had a specific gravity of 1015—1020, and on standing threw down a very copious deposit consisting of a little amorphous phosphate, but mostly of small octahedral crystals of calcium oxalate. Even on superficial examination it was evident that the amount of oxalate present could not be caused by mere deposition, but must be the result of great over-production.

The urine, even that passed after rest, was reddish in colour, and a red film covered the white deposit; blood-reaction was always to be obtained and red blood-corpuscles detected by the microscope. After movement, however, there was pronounced hæmaturia. The urine

contained albumin, but at this time it could not be determined if this were due entirely to the hæmaturia or independent of it. A few ureteral cells and leucocytes were found in the deposit. The right kidney could be palpated and pressed without causing pain, but there was marked tenderness along the course of the ureter, this being accentuated to actual pain at a well-defined spot in the right iliac region. No distension of the pelvis of the kidney could be detected, and there was neither pain nor tenderness on the left side. At this stage of the case examination by the bowel gave no evidence of abnormality at the base of the bladder.

The patient complained of feeling anxious and worried even when not in pain, and his sleep was short and disturbed, beyond the necessary awakenings caused by frequent micturition. He had general aching and vague pains in the limbs, and occasionally in other parts.

A diagnosis was made of (*a*) oxaluria from excessive production of calcium oxalate, this in turn being the result of disordered metabolism caused by the dyspepsia; and (*b*) an oxalate of calcium calculus, lodged in the right ureter. The symptoms and subsequent course of the case, I think, leave no doubt of the correctness of this diagnosis, even though the stone had not been detected by the X-rays. It must be remembered that an oxalate stone is usually small and produces symptoms in severity out of all proportion to its size. It is therefore easily missed by the radiographer, especially if situated low in the ureter, and the one small stone which had already been passed had, indeed, escaped his notice.

At our first consultation I hoped that the blood and pain might be caused by scratching of the ureter during the passage of the oxalate stone recently discharged, but the severity and persistence of the symptoms soon negated this view.

Believing that the oxaluria and atonic dyspepsia were due to a state of low vitality and deficient oxidation, I prescribed absolute rest, as nearly as possible in the open air, with a liberal diet of white meat twice or thrice a week, a little champagne, and a medicinal course of dilute nitric acid and strychnine, with a tabloid of mixed digestive ferments at the beginning of each meal. An aloes and nux vomica pill each night and a dose of Carlsbad salts once a week were ordered to regulate the bowels.

The symptoms, apart from those of calculus, very soon lessened in severity. The appetite improved, the flatulence after food gradually disappeared, the stomach resumed its normal size, and splashing was

no longer to be obtained in the gastric area. The colon still remained distended with gas, and generally, at our consultations, splashing could be elicited over the cæcal area. The large bowel was clearly relaxed, while the stomach had resumed its tone. Almost *pari passu* with the improvement in digestion the deposit of calcium oxalate from the urine lessened, until at the end of four weeks it was comparatively small in amount. At this time the digestion appeared to be normal, and the colon, too, was no longer distended, though constipation persisted. The symptoms of stone, however, were as severe as before, the only difference observable being in the site of the severe pain. This was now deeper in the pelvis, and on rectal examination a distinct resistance could be detected to the right side of the base of the bladder, this spot also being tender. Palpation along the ureter brought out only tenderness where formerly it had produced pain. After micturition pain was sometimes felt at the end of the penis, but usually, as before, the pain radiated into the right testicle and down the inner part of the right thigh. Blood in the urine was constant and increased, like the pain, by the slightest movement, and a constipated motion also caused pain. I formed the opinion that the stone had slipped a short distance down the ureter and had now lodged near the entrance of the ureter into the bladder—almost in the bladder-walls. Copious neutral potations, hot hip-baths, massage along the course of the ureter, and a mixture of belladonna and borax, taken by the mouth, all failed to give any relief. The patient, at my request, made several determined attempts to dislodge the stone by sharp walks, bearing the resulting pain as best he could, but this only made matters worse. It seemed that no treatment could be available other than removal of the stone by an operation, which would probably be severe. The patient, however, wished to avoid this if possible, and yet was desirous of starting on an “exploring” expedition to Mexico early in the present year.

On examining the symptoms of the case, it is, I think, obvious that there must have been some chemical connexion between the gastrointestinal dyspepsia on the one hand and the excessive production and deposition of calcium oxalate on the other, such as that already discussed as being indicated in many cases of oxaluria. With some crude guesses as to the nature of this relationship, I wished to make some experiments on the subject, not with any hope of benefiting the patient, but merely from medical curiosity. Now, there is no “clinical” or easy method for estimating the oxalates of the urine. Neubauer’s method, which I determined to use, is the simplest as it is the oldest, and the numerous

improvements which have been made upon it, while increasing its accuracy, have made it more elaborate and removed it more from the use of the practising physician. But in its original form, with its repeated precipitations and washings and final delicate drying and weighing, some days are required for each estimation, though, of course, several examinations may, with care, be proceeding at the same time. I soon found, however, that such determinations as I thought would be necessary to obtain the desired information would require that the analysis of the urine should be made at least twice, possibly six times for each day—namely, before and after each meal—and not only should the oxalates be estimated, but also the other constituents of the urine, or at least the nitrogenous contents. The composition of each meal also ought to be determined. This is impossible in ordinary practice, nor do I think it could well be done unless two or more persons co-operated in the laboratory work. Probably this is the reason why we are so lacking in precise information as to oxaluria, for I find that observers have hitherto confined themselves to an estimation of the oxalic acid in the twenty-four hours' urine. In the present case, too, the oxaluria and dyspepsia had practically disappeared before I had time to proceed far with such observations as I was able to make, or devise some easier method of arriving at a result, which I think is quite possible. Knowledge of the chemical connexion referred to is of great importance and ought to be capable of attainment. I should certainly attempt the solution again, possibly by simpler means, if another occasion should present itself; but, so far, I have no results worthy of record.

But in making the analysis by the Neubauer method there is to be noted one error of experiment in particular which, though small, must be allowed for if the result is to be even approximately accurate. The acid phosphate of sodium normally present in the urine holds up a certain quantity of calcium oxalate, when, in the initial procedure, the oxalic acid is precipitated by calcium chloride. It therefore seemed to me feasible that if one could increase the acid phosphate of sodium in the urine, by giving large quantities of the salt by the mouth, one might possibly at least dissolve off some of the spikes of the oxalate calculus, and so facilitate its passage. I put this before the patient as an experiment, and he determined to give it a trial before resorting to an operation. He himself had some knowledge of chemistry, and gave me every assistance in collecting specimens of urine at various times of the day. Dr. Robert Hutchison has already shown that the administration of the salt causes considerable increase in the acidity of the urine, but

it was necessary to know whether this was due to the actual presence of the salt in increased amount in the urine. This was ascertained by a trial dose, and will be further discussed in speaking of a later analysis. I ordered at first $\frac{1}{2}$ oz., then 1 oz., and very soon 2 oz. of acid phosphate of soda, to be dissolved in 100 oz. of distilled water and drunk at frequent intervals during the twenty-four hours, but, as far as possible, remote from meal times. For half an hour or so after each dose there was considerable uneasiness in the abdomen, mostly from flatulence, and a feeling of distension; but a little essence of ginger added to the solution relieved this to some extent, and it was never unbearable, considering the end in view. The patient himself tested the acidity of the urine by litmus paper immediately after each micturition, and occasionally forwarded me the whole of the urine passed at each micturition throughout the day. The acidity was increased about half an hour after taking each dose, and the small amount of calcium oxalate still being deposited seemed to be always diminished in the urine passed at this time. This observation, however, is, of course, too rough to be of any real value. The oxalate deposit disappeared entirely in about ten days. The blood in the urine gradually diminished and disappeared in about three weeks. The pain lessened gradually, but in six weeks there was no sign or symptom of stone, the patient could walk ten to twelve miles and take severe gymnastic exercise without the slightest discomfort, nothing abnormal could be detected by the rectum, and he felt quite well and in his usual spirits. On deep pressure over the course of the right ureter there was still a little tenderness, and the urine contained a small amount of albumin, some leucocytes, and a few cells of the ureteral epithelium. This state, I thought, was caused by ureteral catarrh, the result of the scratching of the epithelium during the passage of the spiked stone. No tube-casts were found, and the pulse and heart were normal, thus eliminating kidney disease. Remedies seemed to have no effect upon the condition, so all treatment was stopped and the patient resumed his London work. He now presented himself for life-insurance, putting his whole case before the company, and was accepted—of course at a slightly increased premium, seeing that his urine was not yet quite normal. At the beginning of March he started for Mexico. I had previously taught him how to test the urine for albumin with the magnesium-nitric solution, and he has informed me at frequent intervals of his condition. One of his late communications was that he had spent first three and afterwards four hours in the saddle on the same day, most of the time galloping hard over rough ground. He had in

no way been reminded of his former trouble, and having tested his urine before and after his ride, and also in the interval of rest, he had found no albumin. This healthy condition has been confirmed later on several occasions. It seems to me that there can be no doubt that in this case the impacted oxalate calculus was entirely dissolved by the acid phosphate of sodium. No stone was ever passed by the urethra, and I am sure that not the slightest grit could have escaped the attention of so observant a man. The stone, then, must have been entirely composed of calcium oxalate, which is not a very common condition.

PART III.

This clinical experiment was carried out in the urinary passages themselves, and consequently out of sight. As a control, it was desirable to repeat it outside the body *in vitro* under as nearly as possible the same conditions. An apparatus was therefore devised which is represented in the accompanying plate from a photograph (fig. 1).

A large receptacle contains fluid, which by means of an ordinary mercurial regulator is kept at a constant temperature of 42° C. The fluid is conducted out by a syphon-tube drawn to a point so arranged that between 45 and 50 oz. pass through in the twenty-four hours, and the fluid on leaving the tube was proved to have a temperature varying between 38° and 39° C. The end of the syphon-tube hangs into a test-tube of which the bottom has been removed and the end drawn out into a funnel whose exit is considerably smaller than the calibre of a ureter. Below this a beaker catches the exuding fluid with its contents. The arrangement thus gives an imitation of the passage of urine through a ureter from the kidney, and the beaker represents the bladder. The stone to be experimented upon should be of such a size as, when dropped into the test-tube, to lodge at the mouth of the funnel, and therefore larger than any likely to be found in a ureter. Any diminution of volume of the stone will be better gauged by its gradual descent in the funnel than by guessing its size with the unaided eye.

The fluid to be passed over the stone must be an imitation of the urine passed by the patient after taking a twenty-four-hour dose of the acid sodium phosphate, but it need not contain other ingredients of the urine than the two phosphates of soda, since, so far as we know, none other can influence the solvent action. Indeed the total urine would certainly decompose during a long experiment and would probably foul the stone, the large receptacle, and the tubes, thus causing an intermission of the flow.

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To obtain the exact amounts required of the two sodium phosphates, after the patient had recovered and had for some days ceased treatment, I analysed his twenty-four hours' urine for the respective phosphates, and then, after giving him for two days 1 oz. of acid sodium phosphate daily, dissolved as before in 100 oz. of distilled water, I similarly estimated the phosphates of the urine passed on the second day of the administration.

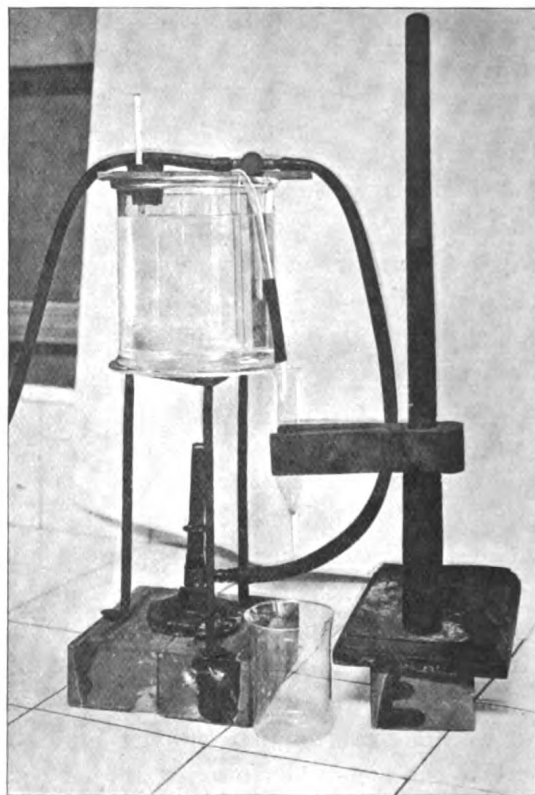


FIG. 1.

Only half his former dose of phosphate was given, for there was reason to think that the former maximum dose was unnecessarily large, and also it was desirable not to exaggerate the amount of salt used in the control experiment. The amount of urine passed on the day in which no phosphate was given was 56 oz., and after the dose of salt it was 95 oz., and the half of this would probably pass through each ureter. The two specimens were examined for total acidity, total phosphates,

and the amounts of the respective sodium phosphates. The acidity was determined by titration with a decinormal solution of sodium hydroxide, using phenol-phthalein as the indicator, and the acidity was calculated in terms of dihydrogen sodium phosphate and also of oxalic acid. The total phosphates were determined by uranium nitrate solution, the strength of which was controlled immediately before the experiment by a standard solution of ammonium phosphate, and the amount of phosphates present was calculated in terms of phosphorus pentoxide (P_2O_5). A 10 per cent. solution of barium chloride was then added to the urine to precipitate the monohydrogen sodium phosphate, and the remaining fluid, which contains the dihydrogen phosphate, was once more estimated for phosphate. The result, subtracted from the total phosphates, gives the amount of monohydrogen phosphate present in terms of P_2O_5 . For convenience, the results of the analysis are given in tabular form :—

	First urine, 56 oz.	Second urine, 95 oz.
Total acidity—		
As dihydrogen sodium phosphate ...	0·408 per cent.	0·78 per cent.
As oxalic acid ...	0·21 ..	0·4 ..
Total phosphates as P_2O_5 ...	0·095 ..	0·3375 ..
Dihydrogen sodium phosphate— ($Na\ H_2\ PO_4$) as P_2O_5 ...	0·047 ..	0·223 ..
Monohydrogen sodium phosphate— ($Na_2H\ PO_4$) as P_2O_5 ...	0·048 ..	0·1145 ..
Total amount in 24 hours of—		
$Na\ H_2\ PO_4$...	0·79 gm.	6·35 gm.
$Na_2\ H\ PO_4$...	0·8 ..	3·26 ..

In these figures there are certain points worth attention before proceeding further :—

(1) The total amount of phosphates passed in twenty-four hours in the first specimen, which is presumably normal urine and which is estimated at 1·59 gm. (24·5 gr.), seems very small when compared with the relative figures given in the text-books, such as 3·5 gm. in terms of P_2O_5 (Neubauer and Vogel) and 48·8 gr. as salts (Parkes). I am certain that the older analyses are all vitiated by the fact that a stock solution of uranium nitrate was used for the analysis. Even recent hand-books give no warning against this error. But a solution of uranium nitrate rapidly changes on keeping, losing its power of precipitating phosphates, and unless its strength be controlled immediately before each experiment by a standard solution of ammonium and sodium phosphate, as was done in the estimations quoted in the table, the phosphates in the fluid to be tested will surely be over-estimated.

(2) As previously pointed out by Dr. Hutchison, the total acidity is greatly increased, in spite of the dilution of the urine. In fact, the percentage of acidity is as nearly as possible doubled.

(3) The acid sodium phosphate administered by the mouth actually appears as such in the urine.

(4) The amount of dihydrogen phosphate in the urine is only a small proportion of that administered. Doubtless much of the salt passed away by the bowels, but some of the difference is to be explained by the impurity of the drug as now dispensed, as will be presently mentioned.

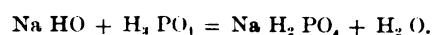
(5) The monohydrogen phosphate of the urine is also greatly increased in amount. This is partly accounted for by the increased amount of fluid washing phosphate out of the tissues, but mainly it must be ascribed to a change in passing through the body of dihydrogen into monohydrogen phosphate.

To ascertain the strength of solution required for the experiment, the percentage of phosphorus pentoxide (P_2O_5) found in the analyses must be translated into terms of dihydrogen and monohydrogen sodium phosphate respectively, and remembering that the molecule of P_2O_5 will form 2 molecules of each of the salts, we arrive at the composition of the required solution as:—

5.35 gm. of anhydrous $Na H_2 PO_4$
 3.25 „ „ „ $Na_2 H PO_4$
 with 95.2 oz. = 1420 cc. of distilled water.

But in attempting to prepare a solution containing this proportion of the respective phosphates, a difficulty arose which brought to light a fact important to pharmacists, and indeed also to those who may prescribe the dihydrogen sodium phosphate. The amounts as estimated necessarily refer to the theoretically pure and anhydrous salts, while those which are dispensed contain water of crystallization. It is easy to drive off this water, but the anhydrous salts are very hygroscopic and difficult to manipulate, while in the process of dehydration there is some risk of transforming a portion of the salts into the pyrophosphate, which has a greatly higher molecular weight. One may allow for the water of crystallization, and there is no difficulty in doing this in the case of the monohydrogen phosphate. This is a pharmacopœal preparation, and must perforce contain 12 molecules of H_2O . The text-books say that the dihydrogen sodium phosphate should have but one such molecule, but it is obvious from the appearance of the salt as supplied by the pharmacists that it must contain a great deal more. I applied to

Mr. Caines, of Messrs. Squire & Sons, for information upon this, and he found that the compound contained at least 20 per cent. H_2O , and probably more, an amount inconsistent with a pure dihydrogen phosphate, and on analysis he found in it some monohydrogen phosphate with an important amount of sodium sulphate. When given by the mouth this mixture of salts would be separated into its several parts, and clearly the total weight of the compound administered would not represent the amount of dihydrogen sodium phosphate desired for the experimental solution; while the sodium sulphate in so large a dose might cause unpleasant symptoms. The monohydrogen sodium phosphate of the pharmacopœia will serve for our purpose when calculation is made of its 12 molecules of water of crystallization, but the dihydrogen phosphate must be prepared in fresh solution by neutralizing phosphoric acid with sodium hydroxide according to the formula—



It is thereby found that the correct proportions ought to be obtained by the following procedure :—

Weigh out 6·6 gramm. of $\text{H}_2 \text{PO}_4$, conc. B.P.

Add 45 cc. of $\frac{\text{N}}{\text{N}}$ Na HO.

Make up to 100 cc. with distilled water.

Dissolve in the liquid 8·2 gramm. of $\text{Na}_2 \text{H PO}_4$, 12 H_2O , and make up to 142 cc.

Each volume of this solution diluted with sufficient distilled water to produce ten volumes ought to give a solution of the same respective strengths of the phosphates as were found in the urine after the administration of the acid sodium phosphate.

As a control the experimental solution was analyzed by Mr. Caines in the same way as the original urine and gave—

Total acidity	= 5·3	gram. $\text{Na H}_2 \text{PO}_4$.
Total phosphates	= 3·337	„ $\text{P}_2 \text{O}_5$.
$\text{Na H}_2 \text{PO}_4$	= 1·775	„ $\text{P}_2 \text{O}_5$.
$\text{Na}_2 \text{H PO}_4$	= 1·562	„ $\text{P}_2 \text{O}_5$.

showing that the solution was as nearly as possible that required.

Mr. Freyer kindly provided me with an oxalate calculus which he had removed from the bladder, the photograph of which appears below. It was larger than any which could probably be found in a ureter, and after drying weighed 0·442 gramm. This was placed in the apparatus and treated by the phosphate solution for six weeks, the same period during which the patient was under similar treatment. The stone was then

obviously smaller, had changed in colour and shape, and had slipped down the funnel into a part much smaller in calibre than a ureter. In the photograph of the apparatus it can be dimly seen. It was of such a size and shape as would be easily passed through the whole of the urinary passages, and possibly even discharged unperceived by an ordinary patient. The solvent process had become very slow in the last few days; after being dried the stone weighed 0.08 gm.

The photographs of the stone before and after the experiment are here given (*see* figs. 2 and 3).

The remnant of the stone was immersed in acetic acid for twenty-four hours to remove any phosphates, but this caused no apparent change. It was then similarly immersed in hydrochloric acid for the same period to remove any remaining oxalate of calcium, and this process caused the stone to shrink to apparently half the size, and, after washing and



FIG. 2.
Calculus before experiment.



FIG. 3.
Calculus after experiment.

drying, it weighed 0.032 gm. The more powerful hydrochloric-acid solvent is, of course, inadmissible in the living passages; but, I believe, that the same great shrinkage would have been effected by a more prolonged action of the phosphate solvent.

The remaining portion of the stone was then powdered. A part treated by liquor potassæ dissolved completely; the remainder dissolved in strong nitric acid on heating, and, after evaporation, gave with liquor ammoniæ the characteristic murexide reaction of uric acid. Clearly, then, we had to do with a uric-acid nucleus upon which the oxalate stone had been built.

It remains to prove that the calcium oxalate of the stone had actually been dissolved, for it might be objected that the stone had only been disintegrated by the 1,365 oz. of fluid which had passed over it, or that the alkali in the fluid had dissolved a uric-acid framework.

To detect oxalic acid in so dilute a solution as that which dripped from the test-tube funnel would manifestly be practically impossible; but the answer to the question is given by two observations.

A certain amount of very fine debris, almost dust, had been washed away from the stone. In the patient this would have been passed imperceptibly from the bladder, but in the experiment the whole had collected in the beaker below the funnel, and had been soaked for six weeks in the test solution. It was now collected on a weighed filter paper. It obviously contained the dark-brown colouring matter of the original stone, and, as it would be difficult to analyze so small a quantity and separate mineral from organic matters, it was determined to weigh it after incinerating to destroy organic matter. The filter paper, with residue, was therefore incinerated on platinum foil, and the weight of the whole, minus the weight of the foil with that of a similarly incinerated paper, gave that of the ash as 0.129 gm. Doubtless some of this is due to the impurities which would fall into the apparatus from the air of the laboratory during a six weeks' exposure; but, even including this item of error, it is obvious that the loss of weight of the stone is not accounted for.

The experiment was repeated with a uric-acid stone actually removed from a ureter, and for which I am again indebted to Mr. Freyer. Before the experiment this weighed 0.32 gm., and it was treated in the apparatus by a similar solution of phosphates; but for this experiment the corresponding salts of potassium were used instead of those of sodium, as being more favourable to the solution of uric acid. At the end of three weeks the stone was paler, but to the eye otherwise unchanged, and it now weighed 0.29 gm. The difference in weight is no more than could be accounted for by the washing away of colouring matter, and it must be assumed that no uric acid was dissolved. Again, for convenience, the results of the experiments are given in tabular form:—

Weight of oxalate stone before experiment	0.442 gm.
„ remnant of oxalate stone after experiment	0.08 „
„ „ „ after treatment with	
A and HCl.	0.032 „
Debris after incineration	0.129 „
Uric-acid stone before experiment	0.32 „
„ „ after experiment	0.29 „

Therefore the thesis is, I think, proved that a calcium-oxalate calculus can be dissolved, both outside the body and in living urinary passages, by acid phosphate of sodium in such a strength of solution as can be produced in the urine of the human subject by the administration *per os* of an easily supportable dose of the salt.

Since in the laboratory experiment successful solution was obtained by such a strength of phosphates as was determined in the urine after the administration of but 1 oz. per diem of acid phosphate, it would seem unnecessary to exceed this amount as a dose, and thus the more disturbing but still bearable effects of 2 oz., which my patient actually took, may be avoided. It would be desirable that a pure acid phosphate of sodium should, if possible, be used, instead of the mixed compound now dispensed as such.

Further, it must be remembered that a calcium-oxalate calculus rarely consists of that salt alone, although in all probability it did so in the case described, thus explaining why no residue was passed. The stone used in the laboratory experiment had merely a uric-acid nucleus, which, when deprived of the surrounding oxalate, would have caused no trouble in the urinary passages. But it is well known that stones occasionally consist of alternate layers of oxalate and of uric acid. It is conceivable, therefore, that in attempting to dissolve a stone one might have to alternate the solvent, removing the oxalate by acid phosphate of sodium, and possibly then dissolving the uric acid by alkaline potassium salts, as recommended by Sir William Roberts.

Finally, I would recommend that in all cases of prolonged deposition of calcium oxalate in the urine occasional doses of acid phosphate of sodium should be given to dissolve the deposit and so prevent the formation of a calculus. If "nervous" dyspepsia were the cause of the condition, the phosphate so given would do no harm; but whether if given continuously it would influence the production as well as the deposition of oxalate is a matter which is worthy of investigation, but on which I have as yet no information to communicate.

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DISCUSSION.

Dr. ROBERT HUTCHISON said he had been very much interested in the paper, for the reason, if for no other, that it seemed to touch upon a subject which was of great interest in pathological chemistry—namely, the acids of the body and their relationships. That was a large subject, but he thought there was none which had more intimate clinical bearings or would prove to be of greater interest to physicians. Any attempt to elucidate it must therefore be very welcome. The paper might be divided into two parts: the theoretical part, which discussed oxaluria and its pathology in general; and the practical part, wherein the author described his case and the method of dealing with the oxalate calculus. The question of oxaluria in general opened up some very large and speculative questions, into which time did not permit him to enter fully. But he was one of that fairly numerous body who were profoundly sceptical about the existence of oxaluric dyspepsia, or the oxaluric "symptom-complex" at all; he believed the creation of that symptom-complex to be a work of the imagination. He believed that oxaluria never produced symptoms, except mechanically, resulting from its separation out in too great quantities from the urine. It was well known that the production of acid in the stomach meant an increase in the alkaline content of the urine, with the consequence that the calcium oxalate became less soluble and was thrown down. This might happen in any case in which an excess of acid was produced in the stomach. Another great difficulty in interpreting so-called oxaluria was that the mere deposition of even a comparatively large amount of calcium oxalate was no proof that an excess of it had been formed. The conditions which were favourable to solution were probably much more complex than ever Dr. Maguire seemed to suppose. He did not touch, for instance, upon the influence of the presence of magnesium salts in the urine, and there were, no doubt, other obscure factors. With regard to Dr. Maguire's case, he thought everyone would admit that he had proved the thesis which he had set out to establish: that by the administration of acid phosphate of soda one tended to dissolve out oxalic calculi. He, the speaker, found some years ago that the acid phosphate was by far the most potent means of increasing the acidity of the urine; more so, by far, than mineral acids. Anything which increased the acidity of the urine would of necessity dissolve and help to keep in solution, oxalate of lime; and so he was not surprised to find that Dr. Maguire, by giving acid phosphate of soda, was able greatly to increase the acidity of the urine, and finally to wash away the calculus. But whether that would be a method of treatment of general application was open to doubt, for if one administered acid phosphate of soda continuously, one would increase the acidity of the urine, and, although this would do good with regard to the oxalates, it would favour the deposition of uric acid. The author thought it might be possible to give acid phosphate until the oxalic-acid layer was cleared away, then

to use alkalies to remove the uratic layers of a calculus. But one did not know when one had cleared away the oxalate layer and got down to the uric-acid layer. Moreover, he did not think it was as safe as some people imagined to interfere with metabolism in that way. Acid phosphate of soda must appreciably reduce the alkalinity of the blood on its route to the kidney; and he did not think that to go on giving something which appreciably lowered the alkalinity of the blood was a procedure certainly devoid of disadvantages. It was conceivable that it might increase the acid dyspepsia, and he had even thought it might facilitate the production of gout, although he must admit that when he was working with acid phosphate of soda and taking it himself, as well as giving it to others, he was never able to find any bad effect, except that in large quantities it tended to produce diarrhoea. He thought Dr. Maguire had been rather unlucky in the preparations of acid phosphate of soda which he obtained commercially. It had been largely used at the London Hospital in cases of ammoniacal urine, and they had no difficulty in getting a substance which was chemically fairly pure.

Dr. BECKETT OVERY desired to mention the observations he had made in examining urines for five or six years. They were purely observations on the urine, because he had not seen the majority of the patients. He could cordially endorse what Dr. Hutchison had said, because, on glancing through his notes before coming to the meeting, he concluded that at least 60 per cent. of people whose urines were examined had a certain amount of oxalate. There were two distinct kinds: the oxalate associated with showers of large crystals, which he concluded were the crystals to which Dr. Maguire specially referred; and a symptom-complex in which there was a large excess of indican in the urine, with a large number of tiny crystals of calcium oxalate. Therefore he thought the importance of calcium oxalate was entirely due to its mechanical effects. From a number of observations which he had made, he was sure people could be perfectly healthy and yet excrete a considerable quantity of oxalate. He asked whether Dr. Maguire had come across cases of albuminuria associated with a large quantity of crystals excreted. Last summer he had under observation the case of a man whose condition had been diagnosed as Bright's disease. He was aged 22, and when the urine was examined he found large quantities of calcium oxalate, but no casts. He looked up the books to see what was best to be done, but could find practically no reference to the matter, and so he put the patient on a mixture of sodium citrate and potassium citrate. He was thus able to reduce the albumin very much, also the amount of the oxalate deposit. He had examined the patient's urine regularly for three or four months, and found the albuminuria very much more marked after exercise. The patient had never had symptoms of stone, and he did not think a calcium-oxalate stone could be in the kidneys. He had been most carefully radiographed. He (the speaker) did not know anything about the chemistry of the matter, but he thought the disappearance of the oxalate must be due to the 60 gr. of citrate three times a day which had been administered. Acetic acid would not dissolve the oxalates; it needed

hydrochloric. He asked in what form the crystals usually appeared; he believed there was a form which was oval. In some urines one found oval crystals, which were sometimes pigmented, but were not uric acid. On one occasion he sent some to Dr. Garrod, who thought them to be calcium oxalate. Since then, at times, he had come across similar crystals. Last night he found a number of them which dissolved in nothing but hydrochloric acid, and he believed they were oxalate crystals.

Dr. NESTOR TIRARD said Dr. Maguire and the profession would deserve congratulation if the conclusions which the author had put forward were proved on investigation to have a firm basis. Oxaluria was one of the most common conditions met with, whether one admitted the author's syndromes, or took it merely as disordered digestion, or as a cause of slight hæmaturia or slight albuminuria. In all those conditions one had met with oxaluria, and it was a condition which had proved difficult to treat. At present he felt somewhat doubtful as to how far the case described by Dr. Maguire could be definitely regarded as one of solution. He thought Dr. Maguire admitted it was possibly not so much solution as disintegration. He did not know that it mattered very much for practical purposes; one was as good as the other, whether it meant the removal of oxalic acid, or whether it favoured the breaking up of any concretions which might have been formed. He had some doubt whether one could grant alternations of storms of uric acid and oxalic acid or calcium oxalate. He understood that Dr. Maguire suggested transformation of one from the other. That appeared scarcely consistent with what was known of the chemical composition of those bodies. Many practitioners had probably been in the habit of using acid phosphate of sodium and sodium phosphate in the treatment of cases of nervous dyspepsia, without, perhaps, any definite thought of its possible action as a solvent of oxalic acid. When he had administered the phosphate of sodium of the Pharmacopœia, usually it had been with more idea of its purgative action than of its action on the urine, and he had employed the salt in that way for a good many years. If the acid phosphate could be used for the diminution of pain in cases of oxalic stone, they would be very much indebted to Dr. Maguire for his observations. But he (the speaker) would like more evidence of cases where the stones had been passed or had been seen by radiography. He admitted that in the present case the evidence of pain over the ureter and the history of hæmaturia appeared fairly conclusive; but pain and hæmaturia might possibly exist without a concretion of any magnitude.

Dr. MAGUIRE, in reply, said he could not doubt the existence of a symptom-complex or, better, a "clinical entity" as defined originally by Begbie under the name of oxaluria, and he thought that the great clinical observers of early times were nearly always right in their large generalizations. But oxaluria did not comprise slight showers of oxalic acid which might alternate with uric acid in the urine, and the term must be limited to a very pronounced condition, where there was so much oxalate of calcium

in the urine as could not be a mere extrusion. Over-production or over-absorption must then have occurred, and he himself believed the condition to be due to over-production from altered metabolism. Metabolism was liable to be interfered with by every meal, and when it had been so far disturbed that the patient was passing large crystals of oxalate of calcium in his urine, and suffering from bleeding and irritation in his kidney, the result of stone, one need not fear to induce change of metabolism in the other direction so long as one did not adopt homœopathic doctrines. Owen Rees, many years ago, held the view that oxaluria was the result of transformation of uric acid into oxalate after passage through the kidneys. He (Dr. Maguire) thought that this could occur, and so account for the rather extraordinary experience of a previous speaker, who found that 60 per cent. of patients deposited oxalate of lime crystals from the urine. On the other hand, an observer had said that only 5 to 6 per cent. of patients in one of the large hospitals of Vienna showed such deposit. He himself thought the relation of those figures was clear, for the urine in the former observations must have stood for some time, and thus decomposed. He doubted very much if the albuminuria which occurs in association with oxaluria depended on irritation by the crystals, for there was but little reason to suppose that crystals existed in the higher urinary passages in many cases. A more reasonable explanation was that the oxaluria was caused by deficient oxidation and deficient vitality; consequently there was a diminished circulation through the kidneys, which he believed was the pathological cause of by far the larger number of cases of so-called physiological albuminuria. Occasionally such cases could be cured, but frequently they could not. With regard to the impurities in the acid sodium phosphate, he had tried three different specimens, and the matter would be further investigated by Mr. Caines and put before the Pharmaceutical Society. There was no question that the acid phosphate of sodium—not the ordinary phosphate—as supplied by the pharmacists, contained vastly more water of crystallization than it ought to do. The so-called normal phosphate, the di-sodium phosphate, ought to have twelve molecules of water of crystallisation, to conform with the conditions of the Pharmacopœia. But the acid phosphate of sodium, or dihydrogen phosphate, was supposed by chemists—not pharmacists—to contain only one molecule of water. Unless further investigation should be followed by its inclusion in the Pharmacopœia, there was no guide as to what allowance must be made in calculating the relationship between the anhydrous and crystalline forms. He thought Dr. Tirard was misunderstanding him as to the two phosphates of soda. He believed that it was Dr. Hutchison who first brought forward the acid phosphate of soda to the notice of the profession as an artificial acidifier of the urine.

Medical Section.

November 23, 1909.

Dr. J. MITCHELL BRUCE, President of the Section, in the Chair.

The Rôle of Fats in the Treatment of Disorders of the Stomach.

By F. CRAVEN MOORE, M.D., and R. L. FERGUSON, M.D.

THE use of fat in the treatment of gastric disorders, not merely as a food-stuff, but with the idea of producing a definite therapeutic effect on the disordered secretory activity of the stomach, is of comparatively recent development, and is based on the recognition of the frequency with which excessive secretory activity of the gastric glands obtains as a pathological condition, and on the demonstration of the depressant action of fats on the activity of normal gastric secretion.

It had long been suspected that the presence of fats in the stomach interfered in some way or other with gastric digestion, a belief which was in some measure confirmed by the observation of Penzoldt, who found that a mixture of coffee and cream yielded a lower acidity of the stomach contents than coffee alone, and more conclusively by the later exact observations of Ewald and Boas, who showed that the addition of bacon fat to the test-breakfast associated with their names resulted in a prolongation of the period of gastric digestion and a marked diminution in the amount of free hydrochloric acid in the stomach contents. As at that period it was the prevalent belief that any deviation from the normal in the functional activity of the stomach must be in the direction of deficiency, and that the subjective manifestations of such deviation depended upon organic acid and gaseous fermentations initiated as the

immediate consequence of deficiency in the antiseptic hydrochloric acid and stagnation of the contents from deficient motor-power, the results of Ewald and Boas served but to emphasize the belief that the administration of fats in gastric disorders, and particularly in such as accorded with the type of acid dyspepsia, were contra-indicated in that they would induce further depression of functional activity and provide a possible source for the noxious acids themselves.

The investigations of the German school, and particularly those of Riegel and his followers, into the secretory activity of the stomach in gastric disorders demonstrated not only that excessive secretory activity of the gastric glands could obtain as a pathological condition, but that such was even more frequent than defective activity, and that in the so-called acid dyspepsia the frequently excessive acidity of the stomach contents was due not to organic acids, but to an abnormally high content in hydrochloric acid itself. This conception of the disorders of the secretory activity has been generally confirmed in its main theses, and in this country, although belief in the acid dyspepsia of organic acid fermentation still lingers, the frequency of pathological hypersecretion is now well recognized. To the brilliant investigations of Pawlow and his co-workers we are indebted for the basis of a more accurate appreciation of the nature and origin of these disorders of functional activity, and also for suggestions as to their rational treatment. The observations of Chigin, Lobasoff, Wirschubsky, Lintwareff, and Sokoloff, in the laboratories of Pawlow, established that the liquid fats, olive oil and cream, were devoid of any excitative influence on gastric secretion when introduced directly into the stomach of the dog, and that their presence exerted an inhibitory influence on the normal energy of the secretory process excited by other food-stuffs.

Following on this demonstration of the depressant action of fats on the secretory activity of the stomach in the dog, certain Continental observers—notably Strauss, von Aldor, Backmann, Akimow-Peretz, Piontkowski, Cohnheim, Walko, &c.—began the administration of liquid fats in such morbid conditions of the human stomach as were associated with hyperacidity of the stomach contents. At first cream and butter were employed by Backmann, then almond oil by Strauss, von Aldor, and Akimow-Peretz, and, lastly, pure olive oil by Cohnheim, Walko, and others. The results recorded were, with but few exceptions—Ewald, Fischl, and Blum—so beneficial in alleviating the subjective discomfort and in improving the nutrition of the patient that the method well-nigh

attained the dignity of an "oil-cure." Our own observations have confirmed in great measure the beneficial results of the administration of liquid fats in conditions of hyperacidity claimed by these previous investigators; they have demonstrated, perhaps more completely than had been previously done,¹ the marked effect of such fats in diminishing the acidity of the stomach contents, and they have opened up some problems for further investigation.

It has been my practice at the Ancoats Hospital to include in the routine examination of such cases as manifested symptoms of gastric disorder (unless contra-indicated) a simple estimation of the secretory activity of the stomach. The method employed consists in the administration first thing in the morning on the empty, fasting stomach of a modified Ewald-Boas test-breakfast, modified to obtain what I regard as being of prime importance for comparative results, constancy of physical character and composition, consisting of 10 oz. of weak tea without milk or sugar, and four breakfast biscuits (Huntley and Palmer) which average about 30 grm. The stomach contents are removed after an interval of one hour, the volume and appearance noted, and in the filtrate the amount of free hydrochloric acid and the total acidity are estimated approximately by titration with decinormal sodium hydrate, solutions of dimethylamido-azobenzol and phenol-phthalein respectively being employed as indicators; the results are stated in terms of the number of c.cm. of decinormal soda solution required to neutralize the respective acidities in 100 c.cm. of the filtrate. The peptic power of the filtrate is estimated by the length of a column of coagulated egg-albumin digested in twenty-four hours at 37.5° C., according to the method of Mett, and in some cases an approximation to the relative content in pepsin itself is made by the dilution method of Schiff and Nierstein.

This systematic investigation of the secretory activity of the stomach in all cases with manifestations of gastric disorder demonstrated the frequency with which excessive secretion obtains, a feature which becomes obvious in the series of control analyses in the following tables, on comparing the values for free HCl and total acidity with the normal values, 20-40 and 40-60 respectively, for such a meal.

With the appearance of Cohnheim's paper on the employment of large doses of olive oil this method of treatment was tried in a few cases, and with such obviously beneficial results, not only in diminishing the

¹ The more recent investigations of Cowie and Munson on some thirty-five cases are in accordance with the results we have obtained.

acidity of the stomach contents, but also on the subjective discomfort and on the general nutrition of the patient, that further investigation of the influence of fats appeared desirable. In some sixty-two cases in which there were subjective manifestations of gastric disorder, either with or without indications of organic disease of the stomach, the effect of the administration of oil of sweet almonds on the secretory activity of the stomach was determined. On consecutive days a plain test-breakfast, and a test-breakfast preceded thirty minutes by 1 oz. of almond oil were given, and in the stomach contents the free HCl and the total acidity were estimated; in certain of the cases the digestive power of the stomach contents (peptic and tryptic) was also determined by the method of Mett. The stomach contents after the oil breakfast separated on standing into three layers: oil on the surface, the fluid contents with its granular sediment below, and between the two a narrow layer of emulsified oil, and but for the presence of the oil the contents were similar in appearance to those after the control-meal. In no instance was there any evidence of the presence of bile-pigments or of variations in the mucus content, and the quantity varied quite irregularly, being in some greater, in others less than in the control.

The results of the analyses (*see table*), expressed in averages where a series of observations were made on any one case, show without exception that the administration of oil is associated with a marked reduction in the acid values, free HCl, and total acidity of the stomach contents. Although it is well recognized that in any individual, normal or otherwise, the acid values of the stomach contents removed at a definite time after a meal of constant composition vary within somewhat wide limits, yet the constancy with which they are diminished after the exhibition of oil in so large a number of cases precludes the possibility of coincidence.

The effect, though constant, presents some interesting variations in degree, not only in the several types of gastric disorder, but also in the same type and the same individual on different occasions. On averaging the results in each of the several series, it appears that the greatest absolute diminution in the acid values after the administration of oil obtains in those which present the highest average acidities with the plain test-breakfast, and, as appears from a comparison of the average results, in the cases of gastric ulcer, duodenal ulcer, and nervous dyspepsia, irrespective of the presence or absence of any lesion of the stomach.

In the individual cases also this is true of the majority ; thus in twelve cases, with a free HCl value of 50 or more, the average absolute diminution of this value is 20, and in all but four the individual diminution ranges from 24 to 35.

In the cases on which a series of observations were made, it was noted that whilst the diminution in the acidity was invariable, it differed in degree on different occasions, and, further—what is of particular interest—the effect of the oil was apparently limited to the period immediately following its administration ; in no instance was there any evidence of its having a more enduring effect on the acid values of the stomach contents.

The averages and also the majority of the individual observations further show that the absolute diminution of the total acidity exceeds that of the free HCl, a result difficult of interpretation, since it apparently depends on a diminution of other factors than the HCl, free or combined (assuming the latter to remain approximately constant with the constant proteid content of the test-meal), which contribute to the total acidity of the stomach contents, and which it might be anticipated would be increased by the liberation of fatty acids in the splitting of the neutral fat by the hydrochloric acid and the possible gastric lipase.

Turning to the peptic activity of the stomach contents, it was found in twelve cases in which observations were made that the digestive power was diminished with the acid values—a result in part, no doubt, of the deficiency in HCl—but also, as shown by the dilution method, of an absolute diminution in the peptic content of the filtrate. This is apparent in all except No. 25 of the following six cases :—

No.	Test meal			Free HCl	Total acidity	Pepsin ¹			Trypsin		
						A	B	C			
25	...	Plain	...	45	62	...	42	27	9	...	0
		Oil	...	15	22	...	2.25	64	12	...	0
26	...	Plain	...	60	78	...	182	144	42	...	0
		Oil	...	32	42	...	81	41	12	...	0
28	...	Plain	...	48	64	...	182	144	30	...	0
		Oil	...	24	33	...	41	38	6	...	0
33	...	Plain	...	28	49	...	72	67	27	...	0
		Oil	...	8	18	...	0	13	1	...	0
34	...	Plain	...	39	55	...	60	81	22	...	0
		Oil	...	17	38	...	25	36	17	...	0
35	...	Plain	...	60	78	...	225	132	36	...	0
		Oil	...	34	50	...	41	84	30	...	0

¹ A, filtrate only ; B, fourfold dilution with $\frac{N}{10}$ HCl ; C, sixteenfold dilution with $\frac{N}{10}$ HCl.

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In three cases the oil was given simultaneously with the test-meal, and resulted in a similar diminution of the acid values of the stomach contents.

The explanation of the phenomena following the administration of fats on a subsequent meal is a problem for the exact methods of experimental physiology rather than for the methods of clinical research. The investigations which have dealt with the matter by the exact methods of Pawlow leave it still in doubt whether the observed phenomena are due to a true depression of gastric secretion or to dilution and neutralization by regurgitant alkaline duodenal contents. Pawlow, from his earlier experiments, in which the introduction of olive oil directly into the stomach of the dog, by means of a sound, resulted in a marked depression of secretory activity, as tested by a subsequent meal of flesh, concluded that fat inhibits the normal energy of the secretory process; that the inhibition is central by reflex stimulation of the inhibitory nerves of the glands or the inhibitory centres of these nerves, and is not a mere mechanical effect of the covering over the mucous membrane and preventing excitation of the nerve-endings.

With the object of testing whether oil might act in some such mechanical way, we made a number of observations in which oil was replaced by petroleum, but in no instance was there any diminution of the acid values. The later experiments of Sokoloff on animals with gastric and duodenal fistulæ showed that the presence of undigested fat in the duodenum suffices to inhibit the secretion of gastric juice, and he concluded that the inhibitory effect of fat originates chiefly from the surface of the duodenal mucous membrane, and not from the stomach. Boldyreff, on the other hand, following up some observations of Damaskin, found that the introduction of oil into the stomach of the dog led to an undoubted flow of bile, pancreatic juice, and intestinal juice into the stomach. Experimenting on himself and one of his associates, he found that the stomach contents, removed one or one and a half hours after the ingestion of 80 c.cm. of a 2 per cent. solution of oleic acid in olive oil, contained trypsin and a lipase of pancreatic origin—a phenomenon which had already been noted by Beaumont in the case of Alexis St. Martin. These investigations have been repeated on the human subject by already a considerable number of observers—Volhard, Faubel, Mohr, Schittenhelm, Levinsky, Molnar, Mahlenbrey, &c.—but with another object—namely, of obtaining via the stomach a

sample of the duodenal contents in order to estimate the functional capacity of the pancreas—and in the majority (70 per cent. to 96 per cent.) of instances it has been found that the administration of oil on the empty stomach sufficed to cause a regurgitation of the duodenal contents into the stomach.

In six observations in which we administered 100 c.cm. of oil on the empty stomach, in two only, where the oil was withdrawn after the lapse of half an hour, was bile-pigment present in the mixture, which was acid in reaction, devoid of HCl in a free state, contained pepsin, and in one case gave a very slight tryptic digestion. In the other four cases the oil was allowed to remain in the stomach for an hour, and in these neither bile-pigment nor trypsin was present in the mixture, which contained a small amount of free HCl and pepsin. Again, as already mentioned, in no instance was the presence of bile-pigments or trypsin noted in the stomach contents after an oil breakfast. Whatever the explanation may be, it remains that the neutral liquid fats administered before food lead to a diminution of the subsequent acidity of the stomach contents; the more so, the greater the tendency of the secretory activity of the stomach to exceed the normal.

Clinically, our experience of the administration of fats, such as cream, butter, almond oil, in conditions in which hyperacidity of the stomach contents has been found to exist, has shown that this action of fats is associated with a very definite amelioration of the subjective manifestations of the condition.

In simple forms of hyperacidity, depending on slight degrees of hypersecretion (the so-called hyperchlorhydria of Riegel), the addition of cream and butter in abundance to the diet, with a diminution of the starches, will often suffice to remove the subjective manifestations. It is only in the more severe types, and particularly the severe degrees, of digestive and continuous hypersecretion so commonly associated with chronic ulcer about the pylorus or duodenum, with heartburn, acid pyrosis, the sudden gnawing pain in the epigastrium ascribed to pyloric spasm, and the vomiting of acid fluid, that the administration of almond oil is undertaken, and then in doses of 1 oz. first thing in the morning, and repeated before subsequent meals if necessary. This method has proved of value in not only alleviating the symptoms which follow the digestion of food in the stomach, but also in contributing very markedly to the nutrition of the patient, and is certainly preferable to the sole use of alkalies. In no case have we found that oil so administered has been

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badly tolerated. The method has proved of such utility, in our experience, that we deem it worthy of more general use.

It is of passing interest to note that if the observations of Boldyreff be well founded, the efficacy of the administration of oil, apart from its nutritive value, would depend on a phenomenon—the regurgitation of the alkaline duodenal contents into the stomach—which has been regarded as obtaining after gastro-enterostomy, and as being, in part at least, responsible for the beneficent effects of that procedure.

GASTRIC ULCER.

No.	Name	Age	Sex	Plain T.B.		Oil T.B.		Difference	
				HCl	T.A.	HCl	T.A.	HCl	T.A.
1	W. M.	48	M.	46	82	37	73	9	9
2	J. S.	33	M.	40	80	37	60	3	20
3	J. F.	32	M.	40	65	31	52	9	13
4	M. M.	37	F.	13	34	Trace	18	13	16
5	L. T.	28	F.	71	95	52	74	19	21
6	M. R.	38	F.	60	90	48	82	12	8
7	E. A.	26	F.	70	100	45	64	25	36
8	H. H.	40	F.	35	51	30	42	5	9
9	A. D.	27	F.	65	90	41	62	24	28
10	J. F.	25	M.	49	70	42	50	7	20
11	A. T.	24	F.	70	89	35	53	35	36
12	M. G.	30	F.	65	90	39	58	26	32
13	C. W.	22	F.	42	60	16	30	26	30
14	E. F.	28	F.	46	65	32	44	14	21
15	A. B.	60	M.	40	71	25	56	15	15
16	J. W.	64	M.	37	86	28	52	9	34
17	T. B.	46	M.	40	58	36	49	4	9
18	M. R.	24	F.	30	56	12	32	18	24
19	C. P.	47	F.	56	78	32	58	24	20
20	M. C.	42	F.	47	64	10	22	37	42
21	J. H.	28	M.	48	66	44	62	4	4
22	C. W.	18	M.	48	74	3	20	45	54
23	M. K.	40	M.	50	74	45	72	5	2
24	J. S.	40	M.	34	60	28	53	6	7
25	S. R.	24	F.	45	62	15	22	30	40
26	J. B.	28	M.	60	78	32	42	28	36

DUODENAL ULCER.

27	F. M.	54	M.	42	65	30	56	12	9
28	J. B.	30	M.	48	64	24	33	24	31
29	G. L.	50	M.	34	54	14	30	20	24

CARCINOMA OF STOMACH.

30	J. M.	67	M.	18	34	10	20	8	14
31	P. L.	48	M.	0	14	0	12	0	2
32	J. D.	67	M.	12	18	0	8	12	10

NERVOUS DYSPEPSIA.

No.	Name	Age	Sex	Plain T.B.		Oil T.B.		Difference	
				HCl	T.A.	HCl	T.A.	HCl	T.A.
33	R. K.	28	F.	28	49	8	18	20	31
34	N. D.	22	F.	39	55	17	38	22	17
35	G. P.	32	F.	60	78	34	50	26	28
36	B. D.	30	M.	32	52	20	40	12	12

DILATATION OF STOMACH (ATONIC).

37	J. W.	58	M.	34	70	10	35	24	35
38	F. F.	19	F.	22	45	18	34	4	11
39	M. G.	57	F.	20	42	15	40	5	2
40	R. F.	38	M.	43	69	30	63	13	6
41	M. J.	59	F.	30	54	10	26	20	28
42	W. B.	42	M.	12	32	Trace	14	12	18
43	M. R.	35	M.	28	46	10	25	18	21

ALCOHOLISM.

44	W. T.	38	M.	27	50	15	32	12	18
45	H. M.	37	M.	58	98	52	85	6	13
46	J. M.	45	M.	16	41	10	34	6	7
47	F. H.	24	M.	28	54	Trace	18	28	36
48	A. F.	39	M.	22	86	10	24	12	12
49	M. K.	40	M.	43	68	32	60	11	8
50	C. J.	32	F.	30	56	13	37	17	19
51	T. A.	54	M.	20	32	?	12	?	20
52	T. C.	60	M.	45	67	39	62	6	5

CONSTIPATION.

53	M. A.	67	F.	20	40	16	32	4	8
54	C. M.	36	M.	14	48	10	40	4	8

MISCELLANEOUS.

No.	Name	Age	Sex	Diagnosis	Plain T.B.		Oil T.B.		Difference	
					HCl	T.A.	HCl	T.A.	HCl	T.A.
55	M. R.	35	M.	Gastritis	28	46	10	25	18	21
56	W. K.	27	M.	Influenza	35	59	29	50	6	9
57	C. K.	55	M.	Aortic disease	35	52	26	46	9	6
58	J. H.	26	M.	Mitral disease	35	68	10	43	25	25
59	J. C.	64	M.	Mitral disease	50	65	18	35	32	30
60	M. B.	37	F.	Severe anæmia	17	42	11	36	6	6
61	G. D.	67	M.	Phthisis	40	57	17	44	23	13
62	M. B.	49	M.	Phthisis	42	62	24	45	18	17

TABLE OF AVERAGES.

Diagnosis	No.	Plain T.B.		Oil T.B.	
		HCl.	T.A.	HCl.	T.A.
Total cases ...	61	38.1	61.2	22.7	42.5
Gastric ulcer ...	26	48.7	72.5	30.5	50
Duodenal ulcer ...	3	41	61	23	40
Carcinoma ...	3	10	22	3.3	13.3
Nervous dyspepsia ...	4	40	58.5	20	36.5
Dilatation of stomach ...	7	26	51	13.2	34
Alcoholism ...	8	31	58.7	21.3	44
Constipation ...	2	17	44	13	36
Miscellaneous ...	8	35.2	56	18	40.5

Note.—T. B. refers to Test-breakfast; HCl, free HCl; T.A., total acidity.

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DISCUSSION.

The PRESIDENT (Dr. Mitchell Bruce) thanked Dr. Moore and Dr. Ferguson in the name of the Section for their very able paper. The Council were very gratified to find the country members coming forward with contributions, and he trusted that this was only one of many similar papers which they might have from the Provinces. The Royal Society of Medicine was most anxious to extend its connexion with the Provinces, and to encourage the present Fellows to come more often to the meetings.

Dr. HERTZ said he was particularly interested because the authors gave a scientific explanation of the excellent results which he had been obtaining by a similar line of treatment during the last three years. He asked whether Dr. Moore's investigations had not extended beyond the administration of the oil before meals. For he thought that in cases of gastric and duodenal ulcer of any severity, particularly where severe hæmorrhage had occurred, or when for any reason there was a question of operation, the simple administration of oil before meals would not suffice to produce a cure. For three years he (Dr. Hertz) had been treating all cases of gastric and duodenal ulcer by means of an exclusively oil diet for the first day or two, and occasionally for longer. The patients took 1 or 2 oz. every three hours, and from the first the pain and tenderness rapidly disappeared. Some of his colleagues at Guy's Hospital, and his friends outside, had tried the same treatment, with equally good results. The exclusively oil treatment had other advantages besides those mentioned by Dr. Moore. It was absolutely non-irritating and it provided the maximum of nourishment in the minimum of bulk—more than double what would be contained in the same weight of dried carbohydrate or protein. This was important, as many patients needed the extra nourishment because they had become so wasted. Even during hæmorrhage it could be safely administered. It had obvious advantages over rectal feeding; the administration of nutrient enemata was accompanied by the secretion of gastric juice, so that gastric juice was present in the stomach without anything to neutralize its acid, whereas when oil was given alone, either little or no hydrochloric acid was produced. It had been said that the administration of oil was all very well on the Continent, but no Englishman would take it. But he knew about twenty-five patients who had undergone the treatment without the slightest difficulty. Some four or five others had refused to take it, but none of the twenty-five had any difficulty after they had once begun. Some vomited the first dose, but this was not repeated. If a patient would not take oil, cream could be given, as that contained a large quantity of fat, and it produced much less hydrochloric acid than did milk. When the diet was gradually increased, an ounce of oil was still given before each meal, and that was a most important means

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of preventing recurrence. If the patient would continue to take a small quantity of oil each day before meals there would be a permanent reduction of the secretion of hydrochloric acid, and the liability to recurrence would be correspondingly reduced. He had given oil before meals in dyspeptic conditions associated with excessive hydrochloric acid, with the same good results as Dr. Moore had mentioned. And he wondered whether the supposed good effect of olive oil in gall-stones was connected with the same thing. No one believed olive oil dissolved the gall-stones, but it sometimes relieved the discomfort. Possibly the discomfort associated with gall-stones was sometimes gastric in origin, and associated with hyperchlorhydria; by diminishing the secretion of hydrochloric acid the oil would relieve the discomfort.

Dr. SAUNDBY said he had been using olive oil in his practice for the last three or four years, and with excellent results; but he had hesitated to try to justify it by any attempt to determine the quantitative amount of hydrochloric acid, because of the uncertain results, from such estimation. But the authors had been able to bring a large amount of evidence to bear, sufficient to convince the sceptic. He had not found it necessary to give such large doses of oil as Dr. Hertz had, and he did not quite appreciate that gentleman's point about the relief of pain. In his own experience, he had always been struck by the absence of pain in cases of gastric ulcer as soon as the patient was put to bed. He had used oil much more for the treatment of hyperacidity, in cases of people who were able to get about and wanted to be relieved. Professor Craemer, of Munich, recommends taking infusion of hops the last thing at night to prevent hyperacidity during the night. Subjects of that condition often awoke at 5 a.m. with hyperacidity. He had used it, and certainly benefit followed, but he was not quite sure whether it was due to the hops. He had used the emulsion of olive oil, making it up with compound tragacanth powder and orange-flower water, which made it pleasant and which patients took without complaint.

Dr. R. HUTCHISON said there could be no doubt, from Dr. Moore's results, that he had proved the effect of oil clinically as Pawlow and his pupils had proved it in experiments on animals. But the chief interest of the paper to him was as to whether it would throw any light on the mechanism by which the oil acted in reducing the gastric secretion. He had had a mind to try the effect of petroleum emulsion, and it was interesting to hear that Dr. Moore had tried it, and found that it had not the same effect as the oil, so that its action could not have been purely mechanical. He therefore supposed it must be a chemical effect. Clinical observation could not be expected to throw more light upon it, and so it would be necessary to resort to the experimental method. The further point was the practical application of the plan. And it was there, in spite of what Dr. Hertz and Dr. Saundby had said, that he remained sceptical; not as to the fact that people if given oil would have their secretion diminished, but sceptical as to the necessity of such a therapeutical

departure. Personally, he did not think there was much difficulty in relieving cases of hyperchlorhydria by simple rules of diet, without putting patients through, what was to many, a repugnant mode of treatment. He had not used it largely in his own practice, but he had encountered a fair number of patients who had been treated in this method by others, and one or two of them had said they preferred to endure their hyperacidity rather than take the oil any longer. One patient got unpleasant eructations afterwards. No doubt patients with hyperacidity had a great repugnance to fats, and perhaps that was why they got hyperacidity. And when they took fats they were apt to get free fatty acids elaborated, than which there was nothing more irritating to the mucous membrane of the stomach. Therefore although the results narrated were very interesting, and bore out Pawlow's work, he was still in doubt as to whether the method was likely to be of very much use in practice. With regard to Dr. Hertz's practice of putting patients upon "pure oil days," he agreed with Dr. Saundby that when once a patient with gastric ulcer was lying in bed with a poultice on the epigastrium, and was put on suitable diet, he or she was not often troubled with pain. Therefore, for the mere relief of pain he did not think Dr. Hertz's plan was necessary. And there were plenty of ways of getting into the patient sufficient nourishment without using such a highly concentrated form as pure oil. Besides, so much fat had other disadvantages. What, for instance, was its effect on the pancreatic secretion? One was apt to forget the pancreas, but would not such fatty food increase the pancreatic secretion unduly? If so, what would be the result if long-continued? Personally, he did not feel tempted to abandon the usual treatment by bismuth and the earthy carbonates. None the less, he thought the Section was much indebted to Dr. Craven Moore for his accurate and laborious investigations.

Dr. CRAVEN MOORE replied that he had not pressed the oil treatment to the same extent as Dr. Hertz had done—viz., placing patients on an exclusively oil diet for one or two days at the commencement of the treatment. He had found it sufficient in the class of case described to give 1 oz. of oil before meals to relieve the symptoms and improve nutrition. In no instance had the patients complained of nauseous eructations after the administration of the almond oil. Almond oil was used in preference to olive oil because it was more palatable than most samples of olive oil obtainable in this country. As further emphasising the utility of the method and the absence of disagreeable features, he cited the cases of two medical men who had been under his care. One had chronic gastric ulcer, but declined surgical interference; the other had duodenal ulcer, for which gastro-enterostomy had been performed, but which had been followed by the return of many of his former symptoms. Both were so enthusiastic about the advantages they derived from the taking of almond oil that they obtained it wholesale in Winchester quarts. He (Dr. Moore) felt that his experience of the method had so far not been

sufficiently great to enable him to dogmatize as to the effect of the administration of oil on the hyperacidity which obtained as a reflex phenomenon in cholelithiasis, but so far as it had gone it seemed to indicate that the administration of oil, and also of alkalies, was less efficient in relieving the hyperacidity of cholelithiasis than that of duodenal ulcer, which it at times so closely simulated. Further investigation might establish this as a method of differentiation of the two conditions. He agreed with Dr. Hutchison as to the efficacy of a suitable diet, the diet composed chiefly of bland fats and proteids, as the fat in the diet not only subserved nutrition, but also diminished the hyperacidity of the stomach contents, and so relieved the symptoms, whilst the protein, in combining the HCl secreted, acted much as an alkali.

Medical Section.

January 25, 1910.

Dr. J. MITCHELL BRUCE, President of the Section, in the Chair.

On the Treatment of Tetanus by the Intraspinial Injection of a Solution of Magnesium Sulphate, with Cases.

By LLEWELLYN PHILLIPS, M.D.

TETANUS, though usually considered a surgical disease, is at the Government Hospital of Kasr-el-Ainy, Cairo, generally treated by the physicians, and, if occurring during the course of a surgical case, the patient suffering is very frequently transferred to the medical side. I think that this procedure is the correct one, for, after all, the major part of the treatment of tetanus is medical, just as is the treatment of most other infective diseases. This, therefore, must be my apology, if apology be needed, for bringing the subject up before you. Now, the case mortality of the disease is very high, and although the disease is comparatively rare in England, yet it is of sufficiently frequent occurrence to demand careful study. In the hotter regions of the globe it is more common, and generally more fatal.

I have collected the cases from the reports of St. Bartholomew's Hospital for the years 1895 to 1907 (the antitoxin treatment having been introduced about 1895), and find that during that period 25 cases were admitted into the hospital with a resulting mortality of 14, or 56 per cent. Had I included the two previous years there would have been 29 cases with 18 deaths, a mortality of 62 per cent.

During the three years 1906-1908, 29 cases of tetanus were admitted into Kasr-el-Ainy Hospital, Cairo, of whom 23 died; but of these, 1 recovered from his tetanus but succumbed to pneumonia. Taking 22, therefore, as the number dying of tetanus, we find a mortality of

75·8 per cent.—a truly appalling figure. (There were 3 other cases admitted during the year 1908, but, as they were treated in the manner described in this paper, they have not been included: 2 died and 1 recovered.) These figures show how common is the disease, and also how fatal it is among the native Egyptians, to whom these statistics apply.

If the cause of death in a series of cases be analysed, it will be found that toxæmia accounts for some, exhaustion from the muscular spasms and insufficient food accounts for others, whilst respiratory failure will in yet other instances be found to be the immediate cause of death. In days gone by treatment was chiefly directed to the relief of the spasms, and, by controlling them, exhaustion and overloading of the system with waste products of muscular action were to a certain extent avoided, and a certain measure of success obtained. Let us, therefore, consider shortly the chief indications in the treatment of a case of tetanus. They are four in number:—

(1) The destruction of the bacilli present and the prevention of further toxic absorption. This is done by the ordinary surgical methods, remembering that mixed infections with pyogenic organisms considerably increase the gravity of the case. But this is not always possible, for the bacilli may be generalized, as in one of Blake's cases, in which post mortem they were recovered from the spleen and from the heart-blood, forming a veritable tetanæmia. Bacelli has employed, with excellent results, carbolic-acid solutions hypodermically; they appear to have some antitoxic effect.

(2) The use of antitoxin. As a curative means this is somewhat disappointing, the toxin being too firmly fixed by the nervous system; but as a prophylactic it is of value. To this the American surgeons testify, they having used it on a large scale after Fourth of July celebrations and during the constructional work on the World's Fair; but even then it sometimes fails, being somewhat rapidly eliminated from the system, and so requiring frequent repetition.¹ In reading reports of cases it is difficult in many instances to know how much has been used, some writers quoting the amount in units, others in cubic centimetres; but, anyhow, the best results seem to have been obtained by massive doses. In my cases the amount has been recorded in cubic centimetres. The Lister Institute preparation has been used by me. For some years I had given up the use of the antitoxin entirely, as I failed to see any benefit from its use; but recently, in conjunction with the magnesium-

¹ For further details concerning this, *vide* Vaillard's paper, a *résumé* of which appeared in the *Medical Annual* for 1909, pp. 54 and 55.

sulphate treatment, I have re-employed it. In a case of chronic tetanus under my care in 1903, in which the disease had lasted about one hundred days and in which there was still rigidity of the abdomen, I thought perhaps a dose of antitoxin would clear up matters; but profuse bronchorrhœa set in, the fluid pouring up from the man's lungs, and death resulted in twenty-four hours. After this I discontinued its use.

(3) The relief of spasms. This was formerly obtained by the use of sedatives, such as chloral hydrate and bromides or such bodies. This end is also obtained by the method I am going to describe.

(4) Conservation of the strength by proper food and sleep—absolute quiet, a darkened room, and abundance of fluid to dilute and flush out the toxin being accessory measures.

Solutions of sulphate of magnesium applied to nerve trunks were found to abolish sensibility and motion, producing in fact nerve block. Further, spinal anæsthesia was found to result from their introduction into the spinal canal; for surgical purposes, however, magnesium sulphate is less suitable than such bodies as stovaine, as the effect is less quickly produced and takes a much longer time to pass off. Meltzer accordingly suggested its use in the treatment of tetanus. Meltzer and Auer performed a number of experiments on monkeys, and found, firstly, that in doses of 0·06 grm. to 0·07 grm. magnesium sulphate per kilogramme weight, anæsthesia was produced; larger doses affected the respiration, and yet larger doses paralysed the respiration, but life could be preserved by artificial respiration; lastly, the heart was affected and the blood-pressure fell. Further experiments showed that even in the hyperexcited cord of tetanus, complete relaxation could be obtained without affecting respiration (doses of 0·08 per kilogramme weight being used). Even the face could be relaxed in some monkeys. Again, though all their monkeys injected with tetanus toxin died, those treated with magnesium sulphate lived longer. (Two monkeys were injected with tetanus toxin only, and four with toxin followed by magnesium sulphate.) Two other monkeys died of an overdose of magnesium sulphate. They noted also a cumulative effect after a series of injections, smaller doses being required in the later injections. In the monkeys used, however, the cord extended down to the sacrum, and some of the solution may have been injected into it, hence the cumulative effect. There is also relatively less cerebrospinal fluid present in monkeys, and so the injections caused more spinal disturbance. In man the fluid is more easily distributed, and a smaller dose per kilogramme weight is sufficient. These experiments proved that amelioration of symptoms and retardation of death result from the use of magnesium sulphate.

That the toxin is not neutralized nor destroyed by the magnesium sulphate solution is proved by Cruveilhier's experiments. He found that a mixture of tetanus toxin and magnesium sulphate produced tetanic symptoms in animals even if it stood for an hour before injection. The action of the drug therefore is palliative, and so its use fulfils indication (3).

My attention was drawn to the method by a short report of a case published in the *Lancet*.¹ I therefore tried it in 7 cases, the details of which follow. Whilst I was in England this last summer I looked up all the literature on the subject. I found that in all 21 cases have been published of which only 9 died, a mortality of 42·8 per cent.—a distinct improvement on former statistics. On analysing these further, there were 8 acute cases which recovered, and 8 acute cases which died; 3 subacute cases recovered, 1 other case recovered, but whether these 2 cases were acute or not is uncertain from the report. (Details are given in the table appended.) In one of these cases the author seems to think that death was due to an overdose of magnesium sulphate. My own opinion is that in other cases an insufficient dose was given. To these 21 cases my own 7 cases form a substantial addition. Of them, 4 recovered and 3 died, the proportion being the same as the other cases.

The following is the technique: A 25-per-cent. solution of magnesium sulphate is carefully sterilized and injected in the ordinary way in the lumbar region, allowing first a little cerebrospinal fluid to escape. This is not always easy to do, owing to the lordosis and opisthotonus. In one of my cases it was necessary to give chloroform in order to inject the fluid, as it was found impossible without it. One cubic centimetre of this is injected for every 25 lb. of body weight: this means about 5 c.c. to 6 c.c. for an adult, and 2 c.c. to 3 c.c. for a child. (The doses I used at first were somewhat less than this.) It is as well, if possible, to prop the patient up a little to prevent the extension of the fluid to the medulla, an accident which might lead to respiratory failure. (This happened in a case recorded by Miller. Artificial respiration was, however, effectual, and the boy eventually recovered.) The result is usually flaccidity of the lower limbs and the abdominal muscles, and often slight diminution of the trismus, the patient feeling more comfortable and sleep often ensues. Anæsthesia, however, may not be well marked. It should be repeated as often as is necessary. In acute cases I have generally repeated it every twenty-four hours, but this depends largely on the return of the spasms, a shorter or longer interval being necessary. In one case as many as

¹ 1907, ii, p. 910.

sixteen injections were used, and I saw no bad results after any of them. I have, in fact, seen no bad results at all, save a rise of temperature after the sixth injection. Some of the later injections were larger, no cumulative effect being noticed. In one of my fatal cases my house physician was doubtful if the injections were really intraspinal, as no fluid escaped, only blood appearing. Unfortunately, the spinal column was not examined post mortem.

The following are the details of my 7 cases, 3 of which, as already stated, were fatal. Therefore, of 28 cases so treated, 12 died—a mortality of 42·8 per cent. :—

(1) A. M. A., a boy aged 7, was admitted to Kasr-el-Ainy Hospital on October 31, 1908, having been run over by a cart on October 29. He had an ulcerated wound on the outer side of the right ankle-joint. On November 5 tetanus developed. The wound was treated with carbolic acid, and 60 c.c. of anti-tetanic serum were given, together with chloral hydrate and bromide of potassium. On November 6 there was no improvement: his legs were rigid; he was injected with a solution of carbolic acid. On November 7 he was transferred to my care: all the treatment was stopped, and on November 8 he had his first injection of magnesium sulphate, about 2 c.c. being given intraspinally, together with a rectal injection of chloral and bromide of potassium. He was also given nutrient enemata, as he was quite unable to swallow. The intraspinal injection was repeated on November 9 at 6 p.m. and again at 11 p.m. On November 10 he was able to swallow, and the nutrient enemata were stopped. He had further injections on November 11, 12, and 13, when the temperature rose smartly, and they were stopped. He improved after the very first injection, relaxation of the abdomen and legs taking place, and the power of opening his mouth and swallowing soon reappeared. On recovery from the attack of tetanus I noticed that the left arm and leg were partially paralysed, and it was some months before he could walk properly; this I was afraid, at first, was due to the injections, but, after interviewing his father, I found that it was of old standing, and was in fact due to old infantile paralysis. This was clinically an acute case and did extremely well.

(2) M. S., aged 27, a baker, was admitted to hospital on December 5, 1908, with a history of tetanus for three days. He had had a small operation performed on November 26. He had a very acute attack of tetanus. He was treated with chloral and bromide, and was injected with magnesium sulphate intraspinally on December 5, 4 c.c. being given, similar doses being administered on December 6 and 7, and 6 c.c. on December 10 (through some mischance he was not injected on December 8 or 9); on December 11 he had 8 c.c., but during the last two days the legs remained rigid, and the spasms were so bad that he had to have chloroform inhalations. He died on December 12; his temperature was 37·4° to 37·7° C. The medical resident who gave the injections was not certain that the fluid really entered the subarachnoid space, as only blood flowed from the needle and not cerebrospinal fluid; however, I have

included the case. It was a very severe case, but life seems to have been prolonged by the treatment; he lived ten days after the first appearance of the disease. (Compare the experiments on monkeys, in which, though no cure resulted, life was prolonged.) Post mortem nothing was discovered. No antitoxin was used in this case.

(3) M. A., aged 26, a labourer, was admitted on November 12, 1908, with acute tetanus of one day's duration. He had trismus and stiff neck, and his was an acute case; he had a contused wound of the great toe. He was given 5 c.c. of the magnesium-sulphate solution, together with chloral and bromide, but no antitoxin. He died early on November 14.

(4) A. A. H., aged 25, was bitten by a camel on January 24, 1909, and admitted to the hospital on January 26, with a compound comminuted fracture of the lower end of the right humerus; this was treated surgically, but necrosis ensued. On March 4 he complained of pain in his molar teeth. On March 5 there was lockjaw and rigidity of the posterior neck muscles, pectorals, abdominal and leg muscles; no rigidity in the arms; he could only separate his teeth 2 mm.; spasms were severe and elicited on the slightest stimulus. He was put into a darkened room, and on March 5, 20 c.c. of antitoxic serum were given, and repeated on March 6. I saw him on March 6 and ordered magnesium-sulphate injections; 2 c.c. were given on March 6, 7, 8, 9, and 10, 2.5 c.c. on March 11, 12, and 13, and 3 c.c. on March 14, 15, 16, and 17. After the injections he felt numbness in his legs; marked relaxation followed in the lower limbs, slight elsewhere. After three injections he could open his mouth better and could swallow easier, and the spasms were much diminished. The length of time of the relaxation increased from about one hour, after the early injections, to six hours, and then the rigidity was not well marked. Spasms were rarely seen. He was re-transferred to the surgeon on March 23, cured of his tetanus. This, though originating so long after the original injury, was a very acute case of tetanus.

(5) E. S. G. A., aged 20, labourer, was admitted with tetanus on March 27, 1909. A month previously he had abscesses on the right ring finger and over the left hip, and seven days before tetanus appeared he opened a small pimple on his back; this formed a sore. Tetanus began six days before admission with trismus and stiff neck. On admission he could not walk, and there was rigidity of the trunk and limbs, with clenched teeth and risus sardonicus; there was a temperature of 39° C., respirations were 26 per minute and shallow, and accompanied by a grunting noise. He had severe opisthotonus and constant convulsions the first two nights in hospital. He was given, on the day of admission, 40 c.c. of antitoxin, the wounds were cleaned with carbolic acid, and 2.5 c.c. of magnesium-sulphate solution were injected; this was repeated on March 28 and 29, in a dose of 3 c.c.; 3.5 c.c. were given on March 30 and 31, and daily till April 11. He had sixteen injections in all. The early injections only affected the lower limbs for a short time; the abdomen was noted as relaxed on March 30, but there was no effect on the jaw. After the tenth injection there

were no more convulsions, and he could open his mouth. He got some bronchitis during the course of treatment, but this was relieved by ammonia and ether. It was throughout a severe case. The dosage of magnesium sulphate was insufficient at first.

(6) H. M. F., aged 12, was admitted on March 12, 1909, with tetanus. No definite history could be obtained; there was, however, a sore on the left heel. On admission he had trismus and risus sardonicus, but he could open his mouth a little; there was some abdominal rigidity and tenderness and some stiffness of the limbs, but he could walk a little; some spasms were seen by a hospital orderly. He was given 50 c.c. of antitoxin on March 13, and 2 c.c. of the magnesium-sulphate solution intraspinally. The intraspinal injections were repeated on March 14, 15, 17, 19, and 21, and on March 22 he could open his mouth well and could eat bread. This was a comparatively mild case.

(7) G. R. A., a coachman, aged 30. Eight days before admission to hospital (August 18, 1909), whilst trying a horse, he fell on the ground and sustained three contused wounds on his leg; the day before admission trismus developed; this was quickly followed by other tetanic symptoms. On admission he was rigid, and had opisthotonus and frequent spasms. The temperature whilst in hospital varied from 37° to 38.5° C. On the day of admission he was given 30 c.c. of antitetanic serum and some chloral and bromide, and a saline enema of 500 c.c. at 12, 4, and 8 p.m. On August 19, 5 c.c. of the magnesium sulphate were given intraspinally at about 10 a.m.; this was very difficult to do on account of the opisthotonus, and chloroform had to be given (the needle had broken in the tissues, and was removed under the anæsthetic) and the wounds cleaned; 50 c.c. more serum were given, and some chloral and bromide. In the evening he was well relaxed and seemed better, but the condition returned, and at midnight he was given another injection of magnesium sulphate. He, however, became worse and died on August 20, about 10 a.m. This was a very acute case.

Thus, out of my 7 cases, 4 recovered, 3 of them being severe cases, especially No. 5. All 3 cases that died were very acute, but it is doubtful in one of them if the injections were really intraspinal.

Short details of the published cases are annexed. In addition there are 3 other cases published of tetanus treated by the subcutaneous injection of magnesium-sulphate solutions. On these latter I offer no comment.

The action of the drug consists entirely in the relief afforded by the diminution of spasms and diminished rigidity.

The treatment is not free from danger; in some of the published cases the authors have even thought that death resulted from the treatment. The principal danger to be feared is respiratory paralysis. This occurred in Miller's case, but after prolonged artificial respiration

the boy completely recovered. Another bad effect that has been noted is bronchorrhœa. This has been very severe in several cases, but in one there were râles in the lungs before its onset, and therefore not entirely due to the magnesium sulphate. I have already mentioned a case under my own observation in which profuse and fatal bronchorrhœa occurred after an injection of tetanus antitoxin in a chronic case of tetanus. I had never before seen such profuse secretion from the lungs. Retention of urine is also common after this method of treatment. Death has also occurred quite suddenly with the patient relaxed, the heart failing before the respiration. This is probably due to the action of the toxin on the heart, as experimentally respiration fails before the heart in magnesium-sulphate anæsthesia. To prevent respiratory failure it is advisable to prop the patient up slightly in bed to prevent the fluid extending upwards to the medulla. Should, however, respiratory trouble supervene, the spinal canal should be washed out with normal saline fluid, and artificial respiration be performed if the symptoms become severe. For bronchorrhœa, Logan has advised the use of atropine hypodermically.

The result of these 28 cases is that 12 died and 16 recovered, a mortality of 42·8 per cent. This is a distinct improvement on former figures, and therefore the treatment is, in my opinion, worthy of more extended trial. If the fatal event can be at all delayed, it is possible that time may be so gained as to allow of the advantageous use of antitoxin, and perhaps death may be averted. In such a disease as tetanus, *rational polypharmacy* has a place.

RÉSUMÉ OF THE PUBLISHED CASES.

(1) POWERS.—A male negro; ten days after a pistol wound of the thigh severe tetanus supervened, convulsions about every minute. He was treated with chloral and bromide, and on the third day 10 c.c. of antitoxin were given. Under chloroform 2 c.c. of the magnesium-sulphate solution were given; at 4.30 p.m. and 7 p.m. there were slight convulsions. The next day he was worse and a bigger dose of magnesium sulphate was given; three hours later he was relaxed. Only these two injections were given. Recovery.

(2) HENRY.—Male, aged 9; first seen on July 28, 1907, six days after a kick from a horse and two days after tetanic spasms started. There were convulsions, rigidity of the jaw and neck, abdomen and legs; he was worse on July 30. On July 31 he was given 3 c.c. of the magnesium-sulphate solution, and in fifteen minutes relaxation commenced; he became drowsy, pulse and respiration increased, and the temperature fell a little. On August 2 again rigid; 3 c.c. more of the solution given; three hours later, drowsy. On August 5, 3 c.c. more were given; after this he was very drowsy and respiration became shallow. No further injections were given and the patient recovered.

(3) HENRY.—Male, aged 19; weight, 123½ lb. Admitted to hospital July 30, 1907, seven days after an injury to the foot. He was quite rigid and had severe spasms. The wound was excised. On July 30, under ethyl chloride, 6 c.c. of magnesium-sulphate solution were given; fifteen minutes later he was drowsy, and slight relaxation was noted. One hour later he was asleep and relaxed. On August 1 clonic spasms reappeared: on August 2 he was worse, with a temperature of 106·8 F., and he died the same day. He only had one injection. The case was very acute and possibly treatment insufficient.

(4) HENRY.—A boy, aged 9, was admitted on August 13, six days after injuring both feet on a nail. Temperature 99·3 F., pulse 96, respiration 24, weight 55 lb. There were râles in his chest. 3.30 p.m., by lumbar puncture, 45 c.c. of fluid escaped, and 3·5 c.c. of the magnesium-sulphate solution were injected under ethyl chloride; this was badly taken. Fifteen minutes later relaxation commenced and was complete in an hour; he slept after this. Nine hours later spasms returned. The next day he was very ill, and 2·2 c.c. of magnesium-sulphate solution were injected. Some turbid fluid escaped from the spinal canal. He became comatose and died in the evening, with a temperature of 106° F. This was a very acute case with chest complications, and seemed hopeless from the first.

(5) HENRY.—Male, aged 45; admitted August 25, 1907, having injured his foot three weeks before. There was trismus and rigidity of the abdomen; it was apparently a slight case. On August 27 he was worse. On August 30 he was given 18 c.c. of antitoxin. On August 31 he was almost comatose, with râles in his chest; he was given 30 c.c. more antitoxin. On September 1 he was sleeping like a drunkard, and his temperature was 106·6 F. On August 27 he was given 6 c.c. of the magnesium-sulphate solution; he immediately felt a burning pain in his spine, which ran up to the base of the skull; in fifteen minutes relaxation had commenced. On August 30, under ethyl chloride, 35 c.c. cerebrospinal fluid were removed and 6 c.c. solution injected. He died on September 1. Chest complications were a feature of this case.

(6) HEINECK.—Male, aged 17; admitted October 22, 1908, having wounded his foot with a rusty nail eight days before; tetanus started on August 21. On August 22 he was given 2,500 units of serum, and, later, 4,500 units in the arachnoid and 1,500 units each in the left sciatic nerve and anterior crural nerve. Large doses of antitoxic serum were given again on August 23, 24, 25, 26, 28, and 30, sometimes locally and sometimes intraspinally—in all, 56,500 units. Magnesium-sulphate solution was given in doses of 5 c.c. on August 23, 25, 26, 28, and 30; improvement was noted after each injection. Cured.

(7) BLAKE.—Male, aged 15, weight 115 lb., crushed three fingers seven days before admission. The wound was disinfected on the third day of the disease. 40 c.c. of antitoxin were injected into the cervical region of the spine and 20 c.c. into the medio-cephalic vein, and in the evening 20 c.c. into the medio-basilic vein. On the fourth day of the disease he had 35 c.c. intraspinally. On the twelfth day after the injury he was given 4·5 c.c. of the magnesium-sulphate solution; this was repeated thirty-three hours later, and

again in another thirty-seven and a half hours he had 8 c.c. of a 12·5-per-cent. solution of magnesium sulphate; this was repeated twenty-seven hours later, and six days later—five injections in all. He recovered. The injections had a marked effect in restraining the convulsions and in relieving pain. Occasional doses of morphia and chloral and bromide were given.

(8) MALLOE (quoted by Blake).—A boy, aged 4, weighing 40 lb., sustained a wound of the skin and subcutaneous tissues of the right leg; this was followed by sloughing. He received four injections of 5 c.c. of antitoxin into the buttock, external jugular vein, spinal column, and back respectively, and occasional doses of morphia and chloral. He had one injection of 1·5 c.c. of the magnesium-sulphate solution. He died twenty-eight hours after the first symptom. *Tetanus bacilli* were recovered from the wound, spleen, and heart-blood.

(9) LOGAN.—A boy, aged 11, weighing 80 lb., had a gunshot wound of the hand eight days before. The wound was cleaned after the development of tetanus; and 50 c.c. of serum were given intraspinally, with sodium bromide and chloral hydrate every four hours. On the third day after admission 10 c.c. of serum were injected into each brachial plexus, each sciatic nerve, and around the wound, making 50 c.c. in all. On the same day, under an anæsthetic, 4 c.c. of the magnesium-sulphate solution were given, and on the fourth day 50 m (not quite 4 c.c.) of the same solution. The anæsthetic did not relax the abdominal muscles, but the magnesium sulphate did. He died forty hours and fifty minutes after the first injection of magnesium sulphate. The heart failed first. There was profuse bronchorrhœa, which yielded to atropine. The convulsions ceased completely after the magnesium sulphate; thirteen hours' rest was obtained. There was some difficulty in introducing the needle owing to the opisthotonus. Temperature (p.m.) 108·2° F.

(10) LOGAN.—A female, aged 24, was vaccinated seventeen days before the onset of tetanus (this was done antiseptically, and she probably became infected later); 100 c.c. of serum were injected thirty hours after the commencement of the disease, and the wound excised. At the same time 4 c.c. of the magnesium-sulphate solution were injected, and repeated seventeen and a half hours later under local anæsthesia. Death took place fifty hours after the first symptom. The magnesium sulphate did not relieve at all; she was moribund at the time of the second injection. This was a fulminating case.

(11) FRANKE.—A man, aged 32, wounded his middle finger twelve days before tetanus commenced. The finger was amputated. Thirty grains of chloral hydrate were given daily *per rectum*. Ninety-one days after infection 1 c.c. of the magnesium-sulphate solution was injected, five days later 2 c.c., four days later 2 c.c. The needle broke in the wound and had to be removed. After each injection the convulsions were relieved, the contraction lessened, and he slept better. He recovered. The dosage was small.

(12) ROBINSON.—A boy, aged 12, weighing 67½ lb., developed, after a contusion of his scalp, an attack of tetanus. The wound was excised, and chloral and bromide given daily. On the eleventh day of the disease 3 c.c. of the

magnesium-sulphate solution were injected under an anæsthetic, on the twelfth day 3.5 c.c., and on the fifteenth day 4 c.c. There was marked improvement under the use of magnesium sulphate, which lessened convulsions. He recovered.

(13) MELTZER and AUER.—A man, aged 35, developed tetanus four days after a wound of the foot. He was given large doses of antitoxin and sedatives, but without relief. He was given an injection of the magnesium-sulphate solution in the dose of 1 c.c. for every 18 lb. of body weight, and two hours before his death antitoxins intravenously. The anæsthetic and relaxing effect of the injection was good, but he died five hours after it. The respiration was good to the end.

(14) MILLER.—A boy, aged 7, lacerated his left hand seven days before. He was given antitoxin daily for fourteen days, in doses of 1500—7000 units; also sedatives for a short time, and copious saline infusions and enemata. He received eleven injections of the magnesium-sulphate solution in thirteen days, approximately 2.5 c.c. each time. "Of the value of the treatment by magnesium sulphate no one who witnessed the case has any doubt." Relaxation lasted eighteen to twenty-four hours and involved all the muscles. Several times there was respiratory collapse lasting eleven to fourteen hours, and the pulse dropped. He recovered.

(15) PECK (referred to in Meltzer's paper).—A man, aged 35, wounded his foot, and developed tetanus four days later. He was given much antitoxin and sedatives. Magnesium-sulphate solution was given in the proportion of 1 c.c. for every 25 lb., but with only moderate effect; then 1 c.c. for every 18 lb. with good effect, complete relaxation resulting. Pulse 120, respiration 22, temperature 102° F. Three hours later he had 60 c.c. antitoxin intravenously, but died suddenly two hours later. The respiration was good to the end.

(16) HESSERT (Harris).—A male had acute fulminating tetanus, and recovered after several injections of magnesium sulphate. No further details.

(17) HESSERT.—A more chronic case; recovered under several injections of magnesium sulphate. No further details.

(18) HESSERT.—Another case; recovered under several injections of magnesium sulphate. No details.

(19) HESSERT.—A small boy had an injection of 3 c.c. of the magnesium-sulphate solution, but died at night of heart failure, with relaxed muscles.

(20) GRIFFON.—A man, aged 18½, received a gunshot wound on September 1, and tetanus followed on September 14. He was given antitoxin and chloral from September 14—19 without any improvement. On September 19—20 he was given 2 c.c. of the magnesium-sulphate solution and he recovered.

(21) RAMOND.—A man, aged 21, injured his foot six days before tetanus developed. The first symptom was on July 29, 1908. He was admitted to hospital August 1. He had chloral and antitoxin daily until August 5, then less frequently. On August 5 he had 6 c.c. of the magnesium-sulphate solution, 4 c.c. on August 8, and 3 c.c. on August 14. He had retention of urine. Recovery resulted.

APPENDIX.

In addition to the cases treated by intraspinal injections, three other cases are recorded in which magnesium sulphate was used subcutaneously. They are referred to in Blake's paper as well as in the original papers. The details are briefly as follows:—

LYON'S CASE.—Male, aged 7; history of tetanus for eight days; symptoms severe on the eleventh day. On the twelfth day 2 dr. of a solution of magnesium sulphate were injected under the skin of the abdomen, and repeated on the thirteenth, fourteenth, seventeenth, and nineteenth days. Cure resulted.

GREELEY'S FIRST CASE.—A male, aged 2; tetanus about ten days. Two drachms of magnesium sulphate in a pint of water were injected under the skin; this was repeated the next day. He also had 7500 units of serum. Cure resulted.

GREELEY'S SECOND CASE.—Male, aged 45; tetanus for four weeks: mild case. Three drachms of a magnesium-sulphate solution subcutaneously. Cure resulted.

I wished just to refer to these three cases without comment.

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DISCUSSION.

The PRESIDENT (Dr. Mitchell Bruce) said it was unfortunate that Professor Phillips could not be present, as it was impossible for him to leave Egypt until June. But he judged from the applause which greeted the reading of the paper that the Section would agree to its thanks being conveyed to Professor Phillips for his contribution. The author claimed no originality for the method which he had described, but he had given a very careful record of facts which certainly appeared to be encouraging; and when the paper was published, practitioners might be induced to try the treatment for a disease which appeared to be so intractable otherwise. He hoped some of those present would be able to detail cases of their own which they had observed and treated in the same way. The paper was also of interest from the point of view of spinal anæsthesia, and perhaps those who had not had the opportunity of using the method in tetanus would at any rate be able to say something on that aspect of the subject.

Mr. C. M. PAGE desired to relate the course of a case which was admitted into St. Thomas's Hospital last Saturday, under Mr. H. B. Robinson, to whom he was indebted for permission to quote the instance. The patient was a man, aged 55, a labourer, who had previously been healthy and in regular work. On January 10 a cart wheel went over his right hand, causing a lacerated wound, which he treated himself for some days, without any special precautions as to cleanliness. Seven days later he was admitted to an infirmary, when the wound was said to be in a very dirty condition, and was treated antiseptically. Eleven days after the injury the man complained of pain in the back and neck, which were found to be rigid, and he could not properly open his mouth. On January 23 he was admitted to St. Thomas's. He was a strongly-built man, weighing about 12 st. There was an ulcer about the size of half a crown on the back of the right hand, covered with yellow slough. The face was drawn and lined, and he could swallow only with difficulty. The teeth could not be separated more than $\frac{1}{2}$ in. Neck and back muscles were rigid and painful, but no opisthotonus was present. The abdominal muscles were stiff, but moved with respiration. The muscular condition of arms and legs seemed normal. Temperature 102° F., pulse 100, of good volume and tension. He was given 30 gr. of chloral at four-hourly intervals, slept fairly well, and took some food. On the following day there was no very marked change in his muscular system, and at 10.30 a.m. he was anæsthetized with chloroform and the spinal theca punctured in the second lumbar space, 5 c.c. of cerebrospinal fluid being withdrawn and 5 c.c. of a 25-per-cent. solution of crystalline magnesium sulphate injected. About an hour later he had sufficiently recovered from the anæsthetic for his symptoms to be observed: he was conscious, and there was a flaccid paralysis of both legs; the knee-jerks, cremasteric and abdominal reflexes were absent, though the triceps-jerks were present, and

the pectorals reacted to cutaneous stimulation. The abdomen was soft and moved fully with respiration, and voluntary movement was present in the arms. His colour was not good, but he could swallow more readily than before the puncture was done. At 3 o'clock he stopped breathing, but the pulse continued strong, and, after a few movements of artificial respiration, spontaneous breathing recommenced. After the injection of the magnesium sulphate he was not propped very high in bed. At 5.30 his arms were found to be flaccid. There was practically no movement in the chest, and the neck muscles were relaxed. The man was cyanosed and quite unconscious. He (Mr. Page) therefore punctured the theca in the second lumbar space, and cerebrospinal fluid trickled out, but not under much pressure; 6 c.c. of cerebrospinal fluid were withdrawn and kept for examination. Respiratory failure occurred at this stage, so he injected 5 c.c. of normal saline, containing $\frac{1}{15}$ gr. of strychnine, direct into the subdural space, and artificial respiration was carried out. He started breathing almost at once, and continued steadily to do so for half an hour, though the movements were shallow. The pulse then became weaker, and respiration stopped at 6.30 p.m. The heart also failed, and, in spite of the continuance of artificial respiration for another fifteen minutes, there were no signs of recovery. Nothing remarkable was found post mortem: there was some cirrhosis of the liver and thickening of the pia-arachnoid; the heart was large, but the valves healthy. Histological examination of the nervous system had not been completed. The specific gravity of the cerebrospinal fluid withdrawn on the second occasion was 1009. Chemically it showed traces of magnesium sulphate. The attack of tetanus was not a very acute one, and Mr. Page believed he died directly from the paralysing effects of the injection of the magnesium sulphate. The dose given was in accordance with that laid down by Dr. Meltzer. The dosage in this patient amounted to considerably under 1 c.c. for every 25 lb. of the body weight.

Dr. A. E. RUSSELL said there was no doubt about the influence of spinal anæsthesia in the control of spasms of tetanus, and he had seen it control the spasms of strychnine poisoning in animals. If cocaine were injected into the lumbar spinal theca of a dog, and *m* iiij or *m* iv of the liquor strychninæ of the British Pharmacopæia were then injected, the hinder part of the animal would show no strychnine spasms. But the fore-limb showed the spasms with the slightest stimulation. He thought the meaning of that was that the cocaine did not spread up readily from the lumbar to the cervical region; for if the cocaine were injected into the cervical region, the strychnine spasms did not occur in the cervical region, but could be obtained from the hinder part of the animal. As the cocaine influence passed off, the strychnine convulsions recurred. So that some form of spinal anæsthesia seemed to have a definite future in the case of tetanus, but scarcely for strychnine poisoning, because the patient was usually dead before treatment could be tried. If the magnesium sulphate caused a more prolonged anæsthesia than cocaine or stovaine or novocaine, it would be a better method of causing anæsthesia; but the case recorded by Mr. Page was a very serious one.

Dr. CASSIDY remarked that, in spite of what Professor Phillips said, the injection of magnesium sulphate seemed, in the light of the case narrated by Mr. Page, to be attended by danger; and the effects of novocaine or other spinal anæsthetics were too transient to make them valuable. The rational treatment seemed to be to make an effort to get the tetanus antitoxin into contact with the central nervous system. That had been done by injecting it into the lateral ventricles, but that necessitated an extensive surgical procedure. One's first aim in treating a case of tetanus ought to be to cut off external stimuli as far as possible, and if one made a large trephine hole in the skull much of the good from injecting the serum would be counteracted; but antitetanic serum could be introduced into the spinal theca with but little disturbance to the patient: there was no irritation after the operation, and the procedure was very valuable. He had the charts of the case of a child who came to hospital without any wounds on the body, but with a very foul mouth and carious teeth. She came on the fifth day of her illness, and she had most violent tetanic spasms, with extreme episthotonus and pleurothotonus, and the least stimulus caused convulsions. She was given antitetanic serum on the sixth, seventh, and eighth days, but without effect, and the temperature steadily rose to 101° F., 102° F., and 103° F. On the tenth day he did lumbar puncture, removing 10 c.c. of cerebrospinal fluid, and injected the same quantity of antitetanic serum. The temperature, which was 104.6° F., fell to 101.3° F., with an evening rise to 102° F., and next day there was a steady fall. On the fifteenth day it was normal, and she had had no convulsions after the injection. Everyone who saw the case agreed that the measures had done her much good.

Mr. ARTHUR BARKER said he had had very little experience of tetanus within the last decade or so—only one case, and that recovered on the injection of antitetanic serum through the dura of the skull. The operation was done by a colleague while he (Mr. Barker) was away. He was hoping that someone associated with physiological research would give some explanation as to why it was that a solution of sulphate of magnesium should produce a lowering of sensibility and irritability of the cord and nerves. It might be due to an abstraction of water from the cells of the cord and nerve roots, following on the introduction of a highly hypertonic solution. He froze a 25-per-cent. solution of the substance used in the injections, and he found a reading of -2.7° C. in relation to distilled water. Blood and normal saline freezing at -0.56° C., it was obvious that the magnesium-sulphate solution must be highly hypertonic, and must rob all living tissue cells of water on coming in contact with them. That robbing of the tissues of water was probably a factor in the lowering of the irritability of the cells which existed in the motor tracts. In regard to such injections the position and posture of the patient were important. He believed the specific gravity of the fluid (specific gravity 1.131 since obtained) was much greater than that of the cerebrospinal fluid, which was 1.007, and therefore it ought to be completely under control on adjusting the patient's position, so that its effect could be localized to any particular spot. He did not understand it travelling up to the respiratory centres quite so rapidly as had been indicated in

some of the cases. Experience of spinal analgesia had shown that dense, somewhat viscid, fluids could be located to any particular segment of the cord or any set of nerves. He thought the term "intraspinous" injection should be modified, and he was glad to hear the term "intrathecal" used in the discussion. There was an idea on the part of some people that the spinal cord was injected, but that was out of the question. In that connexion he could not help reflecting on the tolerance of the spinal canal to interference. At one time it was thought to be so exceedingly sensitive, that anything which interfered with it in any way would necessarily end in disaster. But those who had observed cerebrospinal meningitis would know its tolerance was considerable. The same notion was held not long ago as to the extreme vulnerability of the peritoneum, but it was now known to be very tolerant. The cord and nerve roots were exceedingly delicate, and must be treated with profound respect, but they were probably surrounded by defensive agencies to a larger degree than had hitherto been suspected, as was indicated by operative experience, by pathological observation, and by experiment.

Mr. T. P. BEDDOES said there seemed to be one fallacy in the figures put forward that evening, namely—that a greater mortality from tetanus was made out in Cairo than in London. That might be accounted for by the cases in London coming for treatment earlier, because there was little doubt that, although tetanus was vastly more common in hot climates, the mortality was very considerably less. Those who practised in the Tropics, although they had good preparations of tetanus vaccines, were not so frequently in the habit of using them, because the other methods of treatment were, on the whole, fairly successful. And there seemed little doubt, from laboratory experiments, that the application of heat to an animal had considerable effect in warding off the fatal influence of tetanus. Experiment also showed that an animal injected with a measured dose of tetanus and then subjected to a fairly high temperature survived, whereas the control animal did not. Also an animal treated with pilocarpine survived,¹ and that might be the explanation of the two cases from St. Thomas's. The explanation of one of the cases surviving might be that it came under treatment at a warmer time of the year—viz., in March—whereas the fatal case was treated in January. And he submitted, that if the cases which were treated with magnesium sulphate were subjected to a certain amount of treatment which experience and laboratory experiments had shown to be successful, the magnesium treatment would probably be found to yield much better results.

¹ "The experiments of Salomonsen and Madsen have shown that pilocarpine produces in immunized animals a rapid increase in the amount of antitoxin in the blood," Hewlett, "Serum Therapy," 1903, p. 26.

Medical Section.

February 22, 1910.

Dr. J. MITCHELL BRUCE, President of the Section, in the Chair.

Pericardial Effusion: Its Diagnosis and Treatment.

An Address Introductory to a Discussion on the Subject.

By SAMUEL WEST, M.D.

IN opening the discussion on pericardial effusion I am asked to draw attention to the limitations imposed—viz., that it must deal with cases in which the diagnosis is certain and established by *post-mortem examination or operation*. Though this restriction must be strictly observed in the spirit, cases which have recovered without operation can hardly be absolutely excluded, provided *the diagnosis is not open to question*. Still, speakers are requested not to take undue advantage of the licence thus accorded.

I presume that, except in purulent pericarditis, *the effusions must be of considerable size*. *Purulent pericardial effusions may be small, but their early diagnosis is important*. Large pericardial effusions are for the most part *serous*, some are *hæmorrhagic*, and some are *purulent*. Hæmorrhagic effusions are rare.¹

In hæmopericardium, where the effusion is *pure blood*, the hæmorrhage is usually due to *traumatic laceration of the heart or of some large vessel within the pericardium*, or if *consequent on disease* to the bursting of an aneurysm at the root of the aorta, or to rupture of the heart itself.

¹ Twelve cases collected by F. Bryant, *Boston Med. and Surg. Journ.*, 1905, clii, p. 521.

Another group of cases is recorded as met with in the course of *scurvy*, mostly from Russia and East German sources. No instance of this kind, as far as I know, has been reported from this country.

A few cases are recorded in association with *tubercle* or *new growth*, but here, again, I think the cause of the hæmorrhage is to be sought in gross lesions of a large vessel or the heart.

Blood-stained fluid, as distinguished from pure blood, is not so rare, though still rare enough. As in the pleura, it is usually associated with *tubercle* or *new growth*.

With *granular kidney* pericarditis is not uncommon, but usually with little or no effusion, and often with few symptoms. Sometimes the effusion is large. It may contain a considerable amount of blood, or even be pure blood. Hæmorrhagic effusion of any kind in granular kidney is rare enough for every case to be recorded, even if it has been moderate in amount, or discovered only at the autopsy.

For the purposes of this discussion it will be well to focus attention upon the two commonest forms—viz.: (1) Large serous effusion; and (2) purulent effusions, large and small.

SEROUS PERICARDIAL EFFUSION.

These cases may be placed in two groups, *the inflammatory* and *the non-inflammatory* or dropsical. Small amounts of serous fluid are commonly found in the pericardial sac post-mortem, and are probably produced in the process of dying, but in general dropsy the amount may be larger and reach several ounces. Large dropsical effusions may occur in connexion with mediastinal new growth. It is only these that are likely to produce symptoms, and then mechanically owing to their size.

With these exceptions serous pericardial effusion is *inflammatory* in origin, and usually occurs in *the course of rheumatic fever*, to which disease nearly all cases of so-called *primary pericarditis* with serous effusion are to be referred. These rheumatic cases form the most convenient group for discussion, especially as in them the gradual development of the effusion may be watched and studied.

THE PHYSICAL SIGNS OF PERICARDIAL EFFUSION.

One of the earliest results of pericarditis is general dilatation of the heart. The cause of the dilatation is simple. The visceral layer of the pericardium is a thin membrane covering the surface, attached to and

continuous with the fibrous meshwork of the myocardium, which carry the blood-vessels and lymphatics, and within the meshes of which the muscular bundles lie. The inflammation of the visceral pericardium must necessarily involve, therefore, this intermuscular meshwork, and affect the muscular tissue adjacent to it. Thus, with every pericarditis there must be more or less of myocarditis associated, and consequently of cardiac weakness, which will show itself in dilatation. The degree of myocarditis varies, and may be considerable even where the pericarditis itself seems slight, but is certain to be considerable when the pericarditis is severe.

When pericarditis sets in *one of the earliest physical signs is an increase of the cardiac dullness* outwards and upwards on the left side along the third left space or rib. This leads to the disappearance of the cardio-mediastinal notch described by Sibson and named after him *Sibson's notch*. This widening of the cardiac dullness at the left base is often given as a sign of pericardial effusion, but I do not think this is correct, for it is often seen in cases of pericarditis where no other signs of fluid occur, as well as in other forms of heart lesion where there is no pericarditis at all. It is, I believe, *due to dilatation of the left auricle*, which is so much thinner than the ventricle that the same amount of inflammation spreading into it from the pericardium would produce a much greater effect than upon the ventricle, and therefore lead to earlier dilatation.

Another early sign is the disappearance of the cardio-hepatic notch on the right side called *Rotch's notch or angle*. Rotch's notch is difficult to make out satisfactorily in health, even by those who are skilful in percussion. Moreover, it is not constant, and its disappearance is not conclusive of pericardial effusion. In pericarditis it is, I believe, due to *early dilatation of the right auricle*, just like that of the left auricle in the former case.

Both Sibson's notch and Rotch's notch acquired their chief importance from the results of *experimental injections* into the pericardium. These injections were made when the body was lying flat upon the back, but the results of these injections *vary*, as would be anticipated, *with the position of the body*; this has been clearly demonstrated by Aporti and Figaroli.¹ Neither of these signs, then, is of much practical value as a sign of fluid and as distinguishing effusion from dilatation.

Another reason given for both these signs is that the heart sinks in

¹ *Centralbl. f. inn. Med.*, Leipz., 1900, xxi, p. 74.

the serous fluid, so that in the recumbent position it would sink away from the sternum and ribs in these places and allow the fluid to float up into them. It is quite true that the heart when removed from the body full of blood will sink in serum, but it must be remembered that the heart is not quite free to move in the chest, but is attached at the base by the great vessels, and at the diaphragm by the vena cava. When the pericardium becomes distended with fluid the heart does fall away from the front of the chest, but least of all in the places named. The distension of the sac upwards at the base draws with it the big vessels and the heart with them. This is a partial explanation, at any rate, of the rising of the apex beat from the fifth to the fourth space, which is also given as an early sign of effusion. As the distension increases it pushes back the lungs from the front on both sides, right and left, and so the area of cardiac dullness rapidly increases in all directions.

If the dullness downwards and outwards to the left can be shown to extend distinctly beyond the place where the apex of the heart is located, this is one of the most conclusive signs of effusion, but though it is easy to fix by percussion the extent of the pericardial dullness, it is not always easy to fix the exact position of the apex either by palpation or auscultation.

If the amount of fluid be very large indeed, it may thrust the diaphragm down, and the left lobe of the liver with it, so that there may be a distinct bulging or *prominence in the epigastrium in which pulsation may be felt* (*Auenbrugger's sign*). But, on the other hand, in some cases, as in one I recorded, there may be distinct epigastric depression.¹ These epigastric signs, though very obvious when the effusion is very large, cannot be distinguished in smaller effusions, from somewhat similar signs seen where the right side of the heart is greatly distended.

Systolic recession of the epigastrium is sometimes very striking where the effusion is large, and so far may distinguish effusion from dilatation of the right side of the heart, where the systole is usually associated with protrusion and not with recession; but I have seen systolic recession in the epigastrium well marked with adherent pericardium, and I believe it occurs also without either pericardial adhesion or effusion in some cases of dilated right ventricle.

Distension of the pericardial sac by effusion will, of course, produce *great increase in the size of the præcordial dullness*. When a sac becomes distended it tends to assume a globular shape, unless pre-

¹ West: *Med. Chir. Trans.*, 1883, lxvi, p. 252. Clifford Allbutt: *Lancet*, 1883, i, p. 142.

vented in some way. There are three directions where the distension is somewhat restricted—viz., the base where the pericardium has its attachment to the mediastinum, in front where it is in contact with the walls of the thorax, and below where it lies on the central tendon of the diaphragm. The increase, therefore, will be *most marked transversely*, and may be sometimes described as enormous. Thus in a recent case in a young man the dullness reached from 2 in. outside the right nipple line to 4 in. outside the left, and the transverse measurement was about *ten inches*.

The præcordial dullness is often described as *pear-shaped*, and importance is attached by some writers to the shape in the diagnosis between pericardial effusion and dilatation of the heart. Statements differ as to the direction of the *stalk of the pear*, some placing it upwards at the base, others downwards at the apex, others asserting that in the smaller effusions it points upwards, in the larger effusions downwards. As a matter of fact, these descriptions are more or less fanciful and are of no help in diagnosis.

Two other signs are yielded by percussion. The one is called *Bamberger's sign*. This is a small quadrilateral patch of dullness in the left interscapular space. The other is *dullness over the lower lobe of the left lung* posteriorly. This, however, is common enough in morbus cordis where a dilated left auricle presses upon the root of the left lung, or where a greatly dilated left ventricle compresses the lower lobe of the lung directly. Even if these two signs were of any special value they would be of little practical use, as a patient with a large pericardial effusion is, as a rule, too ill to bear the necessary physical examination.

Of the auscultation signs there is something to be said upon the subject of *friction*. It is often stated that the friction which has been present at first disappears as effusion forms, and returns as the effusion is absorbed. This is true in a general way, but requires qualification. It is correct of friction at the apex, but not of friction at the base or along the sternum. It is asserted that it must necessarily be so because friction can only occur when the two layers of the pericardium are in contact, and as in the recumbent position when effusion forms the heart sinks away from the front, so the two layers must be separated, while if the patient be made to sit up and bend forward, the friction which was absent in the recumbent position may become audible again. However, it is a fact that *even in the recumbent position and with a large effusion, friction at the base* may be very loud. Whatever

explanation of this may be given, the fact should be more generally recognized, for too emphatic insistence upon the absence of friction may lead to mistake in diagnosis.

I recall one case in which the diagnosis lay between a large pericardial effusion and a mediastinal new growth involving the pericardium. Decision was given against pericardial effusion because of the loud friction which was heard over a wide area of the præcordial dullness at the base and along the middle of the sternum. Paracentesis was not performed. The patient died, and nothing was found at the necropsy but serous effusion in an unthickened pericardial sac. The life, I believe, might have been saved by paracentesis.

I may remark in passing that double aortic disease with a greatly dilated left ventricle may for a time closely resemble pericardial effusion with friction, though soon the diagnosis becomes clear.

The heart sounds are muffled, faint, or almost absent, as would be expected. Yet this is not conclusive, for as great muffling and weakness may be observed in cases of extreme dilatation.

It may be taken for granted that the *intrapericardial pressure in effusion is considerable*, but I do not know of any manometric observations by which the actual pressure has been measured. In a large purulent pericardial effusion which was opened I have seen the pus ejected from the incision for several inches in a forcible jet.

Fluid under such pressure ought seriously to embarrass the heart, for it would especially interfere with diastole, especially that of the auricles. Consequently we should expect the cervical veins to be distended and to pulsate and a certain amount of cyanosis to be present. The action of the ventricles should be interfered with also, so that the pulse would be small and probably irregular. That, of course, is so. Yet it is extraordinary how little the mere mechanical conditions often seem to affect the heart's action and with how little cardiac distress even a large effusion may be associated. This would suggest that *when the cardiac distress is great there is some other factor* to account for it. This factor is, I believe, the *myocardial change* associated with the pericarditis to which I have already referred.

In all descriptions of the pulse in pericarditis the *pulsus paradoxus*, or *pulsus cum inspiratione intermittens*, is mentioned. Yet in pericardial effusion *pulsus paradoxus* is rare, and when met with is not due to pericarditis or to effusion—i.e., to nothing within the pericardium—but to changes outside the pericardium in the mediastinum—i.e., to what is called *mediastino-pericarditis* or, better, *mediastinitis fibrosa*. The

pulsus paradoxus is not, in my opinion, merely an extreme form of the respiratory curve of the pulse. If it were, it ought to be frequently met with in weak hearts, and it is not. It is due to a band or adhesion so situated that as the chest expands on inspiration the aorta is pinched and the circulation through it so far impeded that the pulse is not produced. Anyway, the true pulsus paradoxus—i.e., where the pulse-beat entirely disappears during inspiration—is a very rare phenomenon in pericarditis.

Empyema *pulsans* is that peculiar form of empyema in which the impulse of the heart is conveyed through the empyema to the chest wall in such a way as to be seen or felt. It might be anticipated that such impulse would be frequent in pericardial effusion. But it is practically unknown. If anything be felt at all it is but a diffuse shock of an indefinite kind, but often even this is absent, except, perhaps, in the epigastrium.

In general symptoms there is nothing characteristic. The patients are sometimes dusky, and may be cyanosed, but extreme *pallor is more frequent than cyanosis.*

Patients prefer the semi-recumbent position on the back with the shoulders raised, the object being, as in other forms of heart disease, to throw the weight off the root of the lungs on to the tendinous portion of the diaphragm, as well as to give free play to the lungs. There is nothing in the appearance of the patient, the decubitus, the dyspnoea, or general condition which can be considered characteristic of pericardial effusion—i.e., nothing which is not met with in morbus cordis or allied conditions.

In respect of the symptoms there is one general statement which may be made—viz., that the *more rapid the development of the effusion, the more severe the symptoms are* likely to be. In other words, a large effusion which has taken some little time to form, so that the heart and lungs have had time to adjust themselves, may be attended with much less distress than a much smaller effusion which has formed rapidly and has not given so much time for compensation. Thus a sudden hæmorrhage into the pericardium may cause death in a few minutes, not from the loss of blood, but from cardiac syncope or shock. On the other hand, even with acute effusion in rheumatic fever, if the effusion has taken some days to develop, as it often does, the symptoms may be much less severe than might be expected, and in some chronic effusions, even of great size, it is the physical signs rather than the patient's distress that attract attention.

DIAGNOSIS.

It is from *dilatation of the heart* generally that pericardial effusion has to be distinguished, for though under ordinary circumstances no confusion is likely to arise, yet between extreme cardiac dilatation and a large pericardial effusion mistakes in diagnosis are by no means unlikely, as the records of paracentesis pericardii show. I have on many occasions been asked to tap the pericardium in cases in which I thought, as the course showed, there was really nothing but dilatation present. To puncture the pericardium is an innocent proceeding, but to puncture a greatly dilated, and therefore thin-walled, heart is very risky, and has often proved fatal. It is not the mere puncture, provided the needle goes right through the wall, but the laceration produced by the movement of the heart upon the point of the needle.

The diagnosis rests entirely on physical signs, for, as has been stated, there is nothing in the general condition or symptoms of the patient which is characteristic, though when the patient has been under observation, so that the development of the effusion has been watched, the diagnosis is easy. *The most conclusive sign is given by percussion*, for though there is nothing characteristic in the shape of the pericardial dullness, still, if the *dullness* can be shown to extend distinctly to the left and downwards *beyond where the apex of the heart* is located, the diagnosis is certain. With dilatation the impulse can usually be made out as far as the dullness extends at the apex, and there is often a *wavy impulse* to be seen and felt in the left intercostal spaces above, unless the action of the heart be extremely feeble, as it may be where the dilatation is extreme. The sounds also, though weak in both cases, may be completely absent with pericardial effusion, except perhaps at the base, while with dilatation they are generally audible to some extent, even down to the apex. *The epigastrium* may be prominent in both cases, but the prominence is more marked in large effusions, and while in pericardial effusion there is often systolic recession, in dilatation there is mostly systolic protrusion. *Time aids largely* in the diagnosis, for though when first seen the dilated heart may be so feeble that its beating is hardly to be detected, it soon recovers itself sufficiently to make the diagnosis clear. As the cases are rarely so urgent as to require immediate operation it is safe to wait awhile, and in a very short time what has been obscure may become plain. I have not myself had the misfortune to tap a dilated heart by mistake for an effusion, but I have often had

difficulty in making up my mind, when the case has first come under my observation, whether I had dilatation or effusion to deal with.

A *mediastinal growth* involving the pericardium may sometimes give difficulty, or a *mediastinal cyst*. Theoretically, an intrapericardial aneurysm may also cause difficulty, but I cannot remember, out of several instances of such aneurysms under my own observation, a single one in which even the suspicion of pericardial effusion was aroused.

X-ray examination might be expected to aid in the diagnosis; it might show more marked pulsation with dilatation than with effusion. With a dilated right heart on deep inspiration a clear space is visible corresponding with Rotch's notch, the cardio-phrenic area. This is said to be absent in effusion. The difficulty of X-ray examination lies in the fact that the patients are too ill to be taken to the apparatus, and its use in the wards on patients in bed is often unsatisfactory.

The *blood examination* in serous pericarditis gives, I believe, no useful information. In the rheumatic cases the changes would be those of rheumatic fever only. But in the case of suspected tubercle the various tubercular reactions might be of some assistance.

THE TREATMENT OF SEROUS PERICARDIAL EFFUSION.

I need not discuss the *general treatment* of pericarditis or pericardial effusion by drugs, counter-irritation, leeches, or other measures, for there is *nothing new* to add. I will confine myself to the consideration of paracentesis.

First, let me say that *paracentesis is rarely necessary*, for serous effusions in the course of rheumatic fever usually disappear spontaneously, and often produce no urgent symptoms. Absorption, when it once begins, is rapid, more rapid than in the pleura. The effusion is, no doubt, removed in the same way, by the lymphatic pump, worked by the respiratory movements on the one side and by the cardiac on the other. Though I have frequently tapped the pericardium, and am always glad to have the opportunity, I must confess that the necessity rarely arises. Even so large an effusion as that to which I have referred disappeared spontaneously, and recovery was complete, so that there are now no signs even of adhesion of the pericardium.

The method is easy and safe with care. An exploration should be made first with a small needle. The needle must be sharp, the hand placed firmly upon the chest, and the puncture made by the fingers rather than by a thrust of the hand and arm. This will avoid anything

like a jump when the needle penetrates the thorax, which might take it too far and possibly injure the heart. The only fatal result I ever saw in a case of paracentesis for serous effusion was due to the laceration of the heart by the sudden jump of the needle. The great secret of safety is to have a *sharp, hollow needle, and to insert it with a gentle, firm pressure*. I have never had any accident myself.

Exploratory punctures may be made *in any place* if the physical signs suggest it. I have tapped, for instance, at the base on the left side and even on the right. In the latter case my puncture withdrew blood only and the withdrawal of the needle was followed by the immediate formation of a blood tumour under the skin the size of a swan's egg. Nothing further, however, happened; the blood was rapidly absorbed and the physical signs which had suggested the site of puncture disappeared.

In an ordinary case, when the diagnosis is clear and there is choice of place given, the sites selected and advocated have been four:—

(1) *In Sibson's notch*—i.e., in the third left intercostal space near the sternum—a dangerous place on account of the proximity of the left auricle.

(2) *In Rotch's angle*, in the fifth right intercostal space, to the right of the sternum—another risky place because the right auricle cannot be far away, even if the effusion be of large size. Sears,¹ in a paper published in the *Boston Medical Journal*, says that he has tapped many times in this position, but generally without obtaining fluid. Schaposchnikoff² says the *third or fourth space on the right side* is better and safer than the fifth, but both are risky. The choice of Sibson's notch or Rotch's angle is based upon the experimental injections which I have already referred to and stated to be inconclusive.

(3) *The fifth space to the left of the sternum*. This spot is advocated because the puncture is made in the spot which is not covered with lung, and so the *pleura* is not perforated. Though this may be a good place for trephining the thorax with a view to incising the pericardium, it is not the best place for paracentesis. Nor need the risk of puncturing the pleura with the needle or with a fairly large trocar and cannula be seriously considered. In paracentesis the mere perforation of the pleura on the way to the pericardium does no harm. I have seen cases in which—where the needle inserted had perforated the pericardium, but the cannula could not be made to penetrate it—the fluid has leaked into

¹ *Boston Med. and Surg. Journ.*, 1906, clv, p. 611.

² *Rev. de Méd.*, 1905, xxv, p. 789.

the pleura and been absorbed rapidly or subsequently removed from the pleura by tapping, in each case with rapid recovery of the pericardium.

(4) The safest place is the *fifth or sixth intercostal space outside the left nipple line* but well within the area of dullness, for here, owing to the displacement of the heart upwards and the distension of the pericardium outwards, is the widest space between the heart and seat of puncture. This is the place I advocate and choose.

All the fluid that can be obtained should be removed. Often it does not re-accumulate, but, if it should, a second and third paracentesis may be performed, or as many as necessary. Cases are recorded in which the *paracentesis has been performed many times.*

Besides the acute effusions which are generally the result of rheumatic fever, there is a group of chronic effusions the origin of which is not so clear ; some are associated with new growth in the mediastinum, or near it ; others, perhaps, with tubercle, though this is rare. Some are without obvious cause : of this the case I have already referred to was an instance ; in this there was a large area of pericardial and mediastinal dullness, which was *thought to be due to a new growth*, because of the loudness of the pericardial friction, and it was on that account not tapped, unfortunately, as it happened, because the necropsy showed nothing but pericardial effusion. *One of the most remarkable cases* of the kind I recorded some years ago,¹ in which paracentesis was frequently performed for what was thought to be probably a mediastinal cyst. The patient lived four and a half years after the first paracentesis and was tapped many times. On his death no mediastinal cyst was found, but only an enormously distended and thickened pericardium.

PURULENT PERICARDITIS.

In purulent pericarditis the effusions are, as a rule, much smaller. When large they yield the same general signs as serous effusions do, and the purulent nature of the effusion, even if suspected, *can only be proved by puncture.* The *smaller effusions are much more difficult* to diagnose, as the pus is often in pockets or pouches, while the rest of the pericardium is adherent. Thus very irregular areas of cardiac dullness are sometimes presented.

In manifest pyæmia the diagnosis of purulent pericarditis may be made on general principles, the heart showing something wrong, which is not due to valvular disease. *Leucocytosis* would only suggest pus somewhere

¹ *Med. Chir. Trans.*, 1883, lxi, p. 248.

in the body but not necessarily in the pericardium, and pericardial effusion in the course of pyæmia is not necessarily purulent any more than pleuritic effusion is. In most cases of pyæmia purulent pericarditis is not diagnosed, either because the symptoms are masked or because there are no definite cardiac symptoms at all. In others the diagnosis may be made of *left-sided empyema*, and on opening the side, after finding or not finding pus in the pleura, the *pericardial sac may be felt bulging*, and incision through the pleural opening shows that it contains pus.

One very interesting and important group of cases is that in which the *purulent pericarditis accompanies or follows pneumonia*. It frequently causes no suggestive symptoms and may be altogether latent. During the acute stage the pericarditis may be very acute and the pericardium be found post-mortem covered with flaky yellow pus, but there is little fluid as a rule and that for the most part sero-purulent.

It is *after the pneumonia* is well over that the most remarkable cases occur. I have more than once, during what appeared to be a normal and satisfactory convalescence, had the patient suddenly die without apparent reason, and post mortem the pericardium has been found full of pus, often with a double empyema. As the physical signs must have been obvious enough, the diagnosis could hardly have been missed if careful examination had been made, so that I now make it an invariable rule, whether there seem reason or not, to *examine a convalescent from pneumonia every two or three days* to avoid being caught napping, as I have been once or twice.

An empyema following pneumonia is generally associated with an irregular temperature curve, but not always, and I *believe that purulent pericarditis is still more often latent* and afebrile. It is specially unfortunate when these cases are overlooked, because just as pneumococcal empyema is of *favourable prognosis*, so I believe is pneumococcal *pericarditis* when recognised and incised.

Purulent pericarditis may become quiescent and the pus inspissate, as many specimens in our museums show.

There is no instance, so far as I know, of the pus in the pericardium spontaneously discharging itself either internally or externally. *If pus points over the pericardium it is an empyema* pointing in a peculiar place, or an abscess connected with the chest wall, but not a purulent pericarditis.

In many cases of latent purulent pericarditis *death is sudden*. This is due to *cardiac syncope*, the result of the *myocardial changes*, which are more extreme with purulent than with other forms of pericarditis.

The examination of the blood is not likely to be of much assistance in diagnosis, but there are not many observations upon the blood in purulent pericarditis recorded.

Of the bacteriology, too, there is much to learn. The pneumococcus has been frequently found in the meta-pneumonic cases, and in the pyæmic cases the streptococcus, staphylococcus, and other bacteria.

On both these subjects we may hope that the present discussion will add to our knowledge.

TREATMENT OF PURULENT PERICARDITIS.

If pus has been shown to be present by exploratory puncture, it must be evacuated either by paracentesis or by incision.

It may seem strange to speak of the *cure of purulent effusion by paracentesis only*, yet this is well known in empyema, and the pericardium seems even more favourably placed for cure than the pleura. Nowadays one never sees an empyema treated by paracentesis, but when the operation for empyema was not so successful in its results, paracentesis was usually tried first and incision only resorted to if paracentesis failed. I have had many cases of empyema cured by paracentesis in my own experience and some of purulent pericarditis, and in the statistics of paracentesis pericardii there are many others recorded.

I recall one case in a girl in which I diagnosed purulent pericarditis, and, after establishing the diagnosis by exploration, I proceeded to introduce a trocar and cannula. However, the instrument was rather blunt, and when I could not penetrate the pericardium without using more force than I thought prudent I stopped the operation, intending to use a sharper instrument the next day. However, in a few hours all signs of pericardial effusion had disappeared, and in its place signs of pleural effusion had developed at the left base. This was tapped then instead, a good many ounces of pus removed, and rapid recovery followed. The pus from *the pericardium had obviously drained out through the puncture into the pleura and had discharged itself into the pleura.*

Nowadays, in suspected purulent pericarditis puncture is usually made for diagnosis, and if pus is found the treatment is incision.

Bearing in mind the fact that the pus in the pericardium may be in a pouch or pocket, and not generally in the pericardial sac, it is well to do what is the rule with a localized empyema: after the pus has been found with the needle, *to leave the needle in, use it as a director or guide*, and follow it down with a knife till the pus is reached.

If, however, the case is of such a kind that free choice of site may be made, there are only two places that need be considered :—

(1) *In the left nipple-line or slightly outside it* within the area of dullness. (2) To the left of the sternum in the fifth interspace.

(1) *The objections to selecting the left nipple-line region* are two :—

(a) That the *left pleural cavity* would be opened. Experience proves this objection is not so serious as theory would suppose, for pneumothorax does not occur, or at any rate collapse of the lung does not, nor if the opening be free does the pus gain access to the pleura. Even if it did, as in the case mentioned as occurring after paracentesis, it is easily evacuated and may be cured by paracentesis. *Hydatids* of the liver have been frequently and deliberately opened from behind through the ribs and pleura without empyema following.

(b) After the incision is made and the pus evacuated the pericardium contracts, and the *tube inserted then runs horizontally* for 2 in. or 3 in. from the external opening. This objection does not seem to be of much importance judging by experience. It does not seem necessary always to retain the tube, for after the pus has been evacuated it does not usually seem to re-form, and the pericardial sac closes in all round the heart, so that in the *course of a day or two the sac is completely obliterated*, as many necropsies show. This is illustrated by the specimen taken from a case in which the side was opened for what was thought to be an empyema. When the finger was inserted the bulging pericardium was felt. This was incised and a large amount of pus evacuated. The case was one of pyæmia, and when the lad died two days later the pericardium was found adherent over the whole heart, and contained no pus.

(2) *The left of the sternum in the fourth or fifth space* is the favourite place among surgeons, and perhaps rightly in those cases in which it is fair to assume that there are no adhesions here, and the pus occupies the whole sac. To be sure of this is, however, just the difficulty. It is not the safest place for exploratory puncture, as has been already stated, and we cannot be sure that the pus is anywhere else except where it has been found by the needle. Operation in this place *may avoid opening the pleura, but not of necessity* ; on the other hand, it involves trephining the thorax there or excision of a portion of the fourth and fifth ribs. This being done, the operation is simple, for the pericardium can be exposed and carefully incised, and the finger introduced into the sac. If nothing be discovered, then the wound can be closed, but the removal of ribs takes time to recover from. Still, it is generally wiser when pus

has been found with the needle to use the needle as a director and follow it down till the pus is reached, and, as most exploratory punctures are made in the left axillary line or thereabouts, the incision is made here too.

Dr. D. B. LEES: When I was asked to take part in this discussion on the subject of "pericarditis with effusion as determined by operation or post-mortem examination," I declined the invitation, because the great majority of my cases of pericarditis have not ended in an autopsy, and because my experience of operation in cases of pericardial effusion has been small. But I was informed by my friend Dr. Voelcker that it was not intended to limit the subject so narrowly, and that it was desired to collect experience on points of diagnosis, prognosis, and treatment. To promote discussion and to save time it will be best for me to pass over the points in which I agree with Dr. West's observations, and to speak only on the points on which my experience seems to have been different from his. I hope that Dr. West will feel that where I differ from him it is not in any spirit of captious criticism, but in furtherance of what I am sure is his desire also—that the subject may be thoroughly discussed, and that the truth may be ascertained with reference to a difficult and important clinical question.

First, I would venture to ask how the statement that "the discussion is limited to cases in *which the diagnosis is open to no question*" is to be reconciled with the statement that "*the diagnosis from dilated heart is often difficult*"? I agree with the second statement, but its truth seems to make the limitation proposed in the first statement quite impossible.

Next, I object to the assertion that the dilatation of heart which accompanies rheumatic pericarditis is "due to direct extension of inflammation to the myocardium." No doubt such extension exists to a greater or less extent, but if it is the cause of the dilatation, how is it that while cardiac dilatation is a marked feature of the pericarditis of rheumatism, it is very slight or absent in suppurative and in tuberculous pericarditis? And how are we to explain its existence in rheumatic cases, often to a considerable amount, without any pericarditis at all? Twelve years ago—in 1898—in a paper read before the old Society,¹ I pointed out that dilatation of the left ventricle is "a frequent, almost a constant, occurrence in a rheumatic attack," and I submitted a series of cardiac tracings taken by myself in proof. The paper was followed by a second paper, by myself and Dr. Poynton,

¹ *Med.-Chir. Trans.*, 1898, lxxxi, p. 401.

in which a further series of tracings, taken by him, from the hearts of children, showed that the statement is true for children as well as for adults. Subsequent experience has led me to be sure that more or less acute dilatation of the left ventricle invariably occurs in acute or subacute rheumatism. It may be very considerable even when there is no pericarditis and little or no endocarditis. In rheumatic pericarditis it is always present in marked degree, and is the chief cause of the great enlargement of the cardiac dullness. The amount of fluid in the pericardium in rheumatic pericarditis is usually much less than is generally assumed; it may even be absent altogether, though the præcordial dullness is large. In an analysis of the post-mortem records of 150 cases of rheumatic heart-disease in children, the great majority of which showed the existence of past or present pericarditis, Dr. Poynton found that in only 12 out of these 150 cases of fatal rheumatic fever was there more than 2 oz. of fluid in the pericardial cavity, and in only 6 cases more than 3 oz. On the other hand, hypertrophy of the heart was noticed in 58 cases, and dilatation was specially mentioned in as many as 92, and in 56 of these it was evidently a striking feature. It is clear, therefore, that the cardiac dilatation which accompanies rheumatic pericarditis is mainly the result, not of the pericarditis itself, but of the morbid condition which is causing the pericarditis. The facts just stated make it clear that experimental injection of fluid into the pericardium of the cadaver can throw no light whatever on the physical signs of rheumatic pericarditis. Such injections were first undertaken on the assumption that the increased præcordial dullness was caused merely by fluid in the pericardial cavity. Now that we know that the dullness is mainly due to dilated heart, we see that such experimental injections are entirely misleading.

Next, I must protest against the statement that the "most characteristic sign of pericardial effusion is the extension of dullness beyond the apex-beat." On careful percussion even of a normal heart it may easily be discovered that the dullness always extends a little way beyond the position of the cardiac impulse. In a dilated heart, as in the case of subacute rheumatism without any pericarditis, the dullness may extend from $1\frac{1}{2}$ in. to 2 in. beyond the position of the impulse. No doubt a considerable extension of the dullness beyond the impulse may in some cases be due to the presence of fluid in the pericardium, but it may be due merely to cardiac dilatation, and therefore it is not even a reliable sign of effusion. And here I would point out that, though in the paper the effusion of rheumatic pericarditis is described as "serous,"

it is in reality sero-fibrinous, and often almost wholly fibrinous with very little serum.

With regard to the treatment of rheumatic pericarditis, I very strongly urge that by far the best local treatment is the persistent application of an ice-bag, while the feet and legs are kept warm by hot-water bottles. If there is much distension of the right heart, as revealed by careful percussion in the fourth and third right intercostal spaces, this should be preceded by the application of leeches between the right nipple and the costal margin, or by a small venesection. A patient suffering from rheumatic pericarditis under my care while Dr. Willcox was my house-physician was bled by Dr. Willcox. Great relief followed, and the use of ice and of salicylate completed the cure. Seven years afterwards he wrote to Dr. Willcox to say that he was still quite well, that he had not missed a day's work since his illness, and that he ascribed his recovery to the venesection.

As to treatment by drugs, my experience has convinced me of the pernicious error of the traditional teaching that sodium salicylate is a cardiac depressant, and therefore to be avoided in pericarditis. On the contrary, a rheumatic pericarditis is one of the most malignant forms of rheumatism, and requires the use of sodium salicylate in large and increasing doses. This is made possible by the addition of twice as much bicarbonate to each dose. In a paper on "The Effective Treatment of Acute and Subacute Rheumatism" which I read before the Therapeutical Section in December, 1908,¹ I related the case of a boy suffering from rheumatic pericarditis, endocarditis, cardiac dilatation, and an enormous crop of large and small rheumatic nodules, cured by salicylate with bicarbonate, the doses being quickly increased from 100 gr. and 200 gr. daily to a maximum of 600 gr. and 1,200 gr. daily. For six days a week for three weeks the remedies were continued at the rate of 500 gr. and 1,000 gr. daily, then gradually reduced. The pericarditis soon disappeared, the cardiac dilatation diminished, every one of his more than 200 nodules vanished, and only a short presystolic and a short systolic apex-murmur remained. I believe that the present ineffective treatment of rheumatic pericarditis is nothing less than a disaster to the patient, and often is the cause of death or of permanent cardiac crippling.

Paracentesis is, I believe, never necessary in cases of rheumatic pericarditis, if treated as I have described. In twenty years' experience at St. Mary's Hospital and fifteen at the Hospital for Sick Children, with constant charge of sixty beds, I have never had recourse

¹ *Proceedings*, 1909, ii (Therap. Sect.), p. 34.

to operative treatment for this condition. Not only is it unnecessary, but it would be extremely dangerous, on account of the great risk of injuring the dilated heart. More than one instance has occurred in which the ventricle has been wounded, sometimes with a fatal result.

The diagnosis and treatment of suppurative pericarditis are much more difficult than in rheumatic pericarditis, and the prognosis is almost hopeless. Not quite hopeless, for occasionally a patient recovers from what is diagnosed as suppurative pericarditis; occasionally a patient with large suppurative effusion is saved by operation; and occasionally one finds in an autopsy a heart encased in a rigid calcareous pericardium, which is probably the final result of a former suppurative pericarditis. I must dissent from the statement that suppurative pericarditis "after pneumonia" is "easy to recognize by physical signs." It may be easy if a large amount of pus is present, but the effusion is often scanty and loculated, and there is then no more difficult problem in physical diagnosis. The difficulty is caused by the fact that the two most marked features of rheumatic pericarditis are absent in suppurative pericarditis. There is little or no cardiac dilatation, and there is a marked absence of friction-sound. I desire to draw special attention to this second fact, because Dr. West does not mention it, and it seems to me of special importance. I have never yet heard a rub in a case of pericarditis which was of suppurative nature. Others may perhaps have had a different experience, but, personally, I have never met with it, though I have been on the look-out for it for twenty years. I am sure that it must be extremely rare, and the fact is of great importance in diagnosis. Some months ago I saw in consultation a man suffering from mitral stenosis and regurgitation, with an enlarged liver and much ascites, who had recently had an attack of pericarditis with marked friction. A very eminent physician had diagnosed this as pneumococcal pericarditis, apparently because there were no joint-pains, and because the pneumococcus and the influenza-bacillus were detected in the sputum, in spite of the fact that one feature of the illness was the existence of frequent profuse acid sweats, and in spite of the history of a severe attack of rheumatic fever twenty-one years before. The recovery of the patient, after the use of tapping, leeches, and hypodermic strychnine, gave the final proof that the former pericarditis had not been suppurative. When I examined this patient the friction had subsided, and the only evidence of his attack of pericarditis consisted in the extension upwards and outwards of the upper limit of the cardiac dullness in the second left intercostal space—a most valuable sign of an early or limited

effusion into the pericardial sac around the great vessels; a sign for which we are indebted to the late Dr. Sansom.

The diagnosis of the presence of suppurative pericarditis in a case of pneumonia—especially in pneumonia of the left lung, which may make it impossible to define accurately the cardiac dullness—may be extremely difficult. It has seemed to me that an indication which is always to be looked for is enfeeblement and great frequency of the pulse. Yet even this is fallacious, for I have seen adult patients recover from pneumonia in which the pulse-rate reached 140.

Dr. WILLIAM EWART regretted that he could not on all points agree with Dr. West. The address itself was a powerful argument in favour of the necessity for diagnosis, for accuracy in diagnosis, and for success in diagnosis; yet the opener did not appear to be a strong believer in their attainment. This might be a sufficient excuse for the criticism of one who stood as a champion for the capabilities of the methods of diagnosis which we possess, and for the hopefulness which lies in their due performance. For the sake of clearness, as there seemed to have been some confusion in reports and quotations from his writings on the subject, he had brought two diagrams (exhibited), so that Fellows might have the information first-hand. In pericardial effusion there were two distinctive percussion signs—one anterior, the other posterior—and, in addition, there were numerous confirmatory signs. These were of value, as it was not possible to foretell that the pericardium in which an effusion took place might not have been of abnormal construction or influenced by unsuspected complications within the chest. This uncertainty might explain the failures in diagnosis to which every physician had to own. If, for instance, there should have been an obliteration of part of the pericardial sac, it was idle to expect that the case could give the normal typical physical signs of fluid effusion. In reality no sign can be said to be *infallible*, not even those to be described as “distinctive.” It was well there were both a posterior and an anterior sign, because one of them could be used with advantage to check the other. He might be allowed to put in incidentally two disclaimers. One was with regard to Dr. Rotch’s sign. Soon before he himself had contributed his observations on pericardial diagnosis, Dr. Rotch had described the valuable sign in question known as “Rotch’s sign.” To refer to it as “Rotch’s angle” was, he ventured to think, to invite further confusion, and therefore undesirable for reasons which he would explain. The sign described by Rotch was the occurrence of dullness at the right

fifth intercartilaginous space. In pericardial effusion, or after artificial injections, this space became dull to percussion. This was an excellent example of a sign which was not distinctive, but only confirmatory; for he had pointed out that this space also became dull in cases of considerable dilatation of the right heart, and that therefore the dullness in question was not in itself able to decide as between a large right auricle and effusion of fluid. A distinctive sign was needed, and the sign he had described as the "*basic angle*" sign was of that kind. If one percussed carefully for the right border of the heart, the outline of cardiac dullness could be traced as a curve working down towards the infrasternal notch. The finding of that curve was the distinctive sign for dilatation. If, on the contrary, the dullness of the fifth space was due to effusion, then its right border would not be curved, but would be found to run obliquely downwards and outwards (instead of inwards), until it was intercepted by the dullness of the liver, in consequence of the shape of the attachment of the pericardium to the central tendon of the diaphragm. The angle thus formed was absolutely distinctive of pericardial as opposed to cardiac enlargement. The basic-angle test was therefore an indispensable adjunct to Rotch's sign. A basic angle could also be made out on the left side for similar reasons. But the apex of the heart being itself angular, the "basic-angle test" was not so striking as it was on the right side. He fully agreed with Dr. West's remarks on the subject of posture as influencing the physical signs. As regards Sibson's diagrams it should be borne in mind that they were the result of percussion for very large effusions or artificial injections rather than for the finer points in the diagnosis of small or moderate effusions; and the question of posture also came in. At our present stage of knowledge, aided by radiography and other tests, we should not rest satisfied with any percussion that was not as accurate as the skiagram itself. In his early days before the discovery of the X-rays he had succeeded by careful percussion in outlining the heart so accurately that the tracings strictly coincided with the outline revealed by the X-rays subsequently.

Posteriorly there existed an equally, if not more, distinctive sign—"the lower dorsal dull patch." But he would first enter another disclaimer in connexion with another sign described by the opener as "Bamberger's sign" in the interscapular space. This might be a very good sign, but he had no knowledge of it, and had not described it.¹

¹ Subsequent to the debate Dr. Ewart realized that the opener's reference was to the well-known *infrascapular patch of tubular breathing and ægophony*, depicted and described as the tenth sign in Dr. Ewart's paper on "Practical Aids in the Diagnosis of Pericardial Effusion," *Brit. Med. Journ.*, 1906 i, p. 720.

The lower dorsal dull patch was remarkable for the constancy of its size and of its shape, which he had been at first puzzled to explain. As a fact this sign, as well as the anterior one, was due to the operation of simple laws of physics. Just as a bag full of water could not be held up without causing its lower corners to fill; it was also impossible to place a bag full of fluid upon a sounding board without interfering with the vibrations of that sounding board. In the present case the sounding board was the liver and the bag of fluid the pericardial effusion. There was a damping of the corresponding part of the liver, over a definite anatomical section of its surface—namely, that under the floor of the pericardium. The lower dorsal dull patch was not on a level with the heart and pericardium, but beneath them, and was merely that part of the liver which supported the weight of the effusion. The test was anatomical and therefore easy to localize, and the dullness, though it was not an absolute dullness, was easily elicited by the pleximeter or by the finger. It should be stated that he had not found the sign quite so easy to estimate in small children, because in their small chest an ultra-careful percusser might obtain the dullness of the lower dull patch even though there might be no fluid, simply on the strength of the dulling effect exerted by the heart itself upon the vibrations of the underlying liver. As to the other signs, they were all simply accessory; but we could not afford to decline their help as there was so much difficulty attaching to diagnosis, and, in particular, to the diagnosis between effusion and cardiac dilatation. In reality both were often present together. The work of Lees and Poynton, following that of Sturges, had made us aware that rheumatic pericarditis was often a pan-carditis. It was therefore most important that signs indicative of fluid should not lead us to conclude that the entire dullness was due to fluid; and that we should not forget the heart itself. Even when there was evidence of fluid, something further was still required—namely, to ascertain how much of heart dilatation there was, and how much of fluid. That was really an examination within an examination. He would therefore urge that there was an additional duty incumbent upon us, that of working up a diagnosis for both effusion and dilatation, which should be not only qualitative, but also, if possible, quantitative. As a fact an examination of the size of the heart through fluid is possible, even sometimes without the X-rays, by simple percussion or by auscultatory percussion aided by posture. A very interesting question raised by the opener was why an empyema should sometimes pulsate and a pericardial effusion not. The following suggested explanation might be capable of

demonstration. The total space occupied by the heart, and the total blood content of its cavities remained constant during all its phases. Its movements did not therefore entail any variation in size and they might not agitate the fluid. It would be quite otherwise if an empyema were in partial external contact with the cardiac surface along which the *waves* of contraction were propagated. Each passing wave might then set up a pulsation in the empyema.

(The discussion was adjourned until March 3.)

Medical Section.

March 3, 1910.

Dr. J. MITCHELL BRUCE, President of the Section, in the Chair.

Discussion on Pericarditis with Effusion, as determined by Operation or Post-mortem Examination.¹

SIR JOHN BROADBENT, in resuming the discussion, remarked that, as a corollary to the title of the paper "On Pericarditis with Effusion, as determined by Operation or Post-mortem Examination," the words "and on the absence of effusion as determined by operation" might well have been added. For he was sure that it must have fallen to the lot of most who had had a large experience of pericarditis to have operated on cases in which effusion had been diagnosed, and to have found instead a greatly dilated heart, with or without an adherent pericardium, but no effusion. The difficulty of diagnosing between these two conditions was often very considerable. His attention was first called to this point in 1893, while acting as house physician at St. Mary's Hospital, when he saw two cases operated on for effusion in which no effusion was found, but only a greatly dilated heart, in one case with the pericardium universally adherent. The first of these cases was that of a girl, aged 7, with a typical rheumatic heart greatly dilated as the result of an attack of pericarditis. The friction rub which had been present for some days gradually disappeared, the area of cardiac dullness increased till it extended to the right nearly as far as the right nipple line, and to the left to 1 in. beyond the nipple line. There was at the same time exacerbation of the severity of the symptoms, the dyspnœa

¹ Adjourned from February 22.

being so extreme that the only position the child could assume with any degree of comfort was sitting up with her arms on a board stretched across the cot and the head resting on the arms. These considerations led to a diagnosis of effusion, and the operation of paracentesis was performed, the needle being thrust in gradually till it penetrated the right ventricle and blood was withdrawn, no effusion being present. She survived the operation eleven weeks, and at the autopsy the pericardium was found to be universally adherent by recently organized adhesions to a greatly dilated heart. The second case was that of an adult, aged 30, in whom the pericarditis was secondary to an attack of pneumonia of the left base. In this case the dilatation was not so extreme, but had increased rapidly, extending $\frac{1}{2}$ in. to the right of the sternum; the friction rub, from being heard all over the pericardial area, had disappeared except at the base of the heart; and the apex-beat could not be seen or felt. The increase in the severity of the symptoms, the pulse-rate being 104 and respirations 40, together with the physical signs described above, was thought to indicate the presence of effusion. The operation of paracentesis was therefore performed, but no fluid was found, the needle entering the right ventricle, from which some blood was withdrawn. The patient seemed none the worse for the operation, but began to improve straightway. The friction rub had entirely disappeared a week later, the area of cardiac dullness gradually diminished till a month later it was approximately normal in extent, and two months later he was discharged cured, being able to walk about and even go upstairs without symptoms of any kind. In this case the pericardium presumably did not become adherent, as the heart entirely recovered from the dilatation.

Since it was now generally recognized that an extreme degree of cardiac dilatation was commonly met with in rheumatic pericarditis, more especially in children, and was frequently associated with general adherence of the pericardium to the heart, whereas effusion to any great extent was comparatively rare, mistakes in diagnosis based on rapid increase of area of the cardiac dullness were less liable to be made. He had failed to find that the shape of the area of cardiac dullness—e.g., the so-called “pear shape” described by Sibson—of any value in determining the presence of effusion. Rotch’s sign was certainly not to be relied on as diagnostic of effusion; in many instances he had found it present in cases of universally adherent pericardium in children, especially in the relapsing cases in which the dilatation of the heart was often extreme.

In his experience effusion to any extent in rheumatic pericarditis was exceptional and rarely met with. By far the most difficult cases, and those in which effusion into the pericardium was frequently missed, were cases of suppurative pericarditis. They were most commonly secondary to suppuration elsewhere—e.g., an empyema: a friction rub was seldom audible throughout their course, and there were usually no specially characteristic symptoms. The absence of a friction rub, and the possible explanation of the pyrexia by suppuration elsewhere, might naturally fail to arouse suspicion of pus in the pericardium, and even when this was suspected the impossibility of mapping out the area of cardiac dullness when this merged into the dullness of an empyema, and the latency of the symptoms, made the diagnosis a matter of considerable difficulty.

When acting as medical registrar at St. Mary's Hospital in 1900 he had seen two cases which were remarkable in that the patients were husband and wife, and that they died within ten days of each other from empyema with suppurative pericarditis. The first was that of the husband, aged 63, admitted to the hospital on April 17. He said he had been well till April 14, when he had a rigor and sudden pain in his chest and then fainted. He became very short of breath, and remained in bed till admitted three days later. On admission he was cyanosed. Pulse, 102; respiration, 42; temperature, 100·8° F. He had a cough, and brought up rusty sputum. On examination, there was dullness over the base of the right lung, back and front, with deficient vocal resonance and fremitus and bronchial breathing; the area of cardiac dullness merged into that of the lung dullness to the right, to the left it extended 1 in. beyond the nipple line. The apex-beat was not visible nor palpable; the heart sounds were distant and feeble, and there was no friction rub audible. He was extremely ill, and died on the third day after admission. There was an empyema at the right base, and the lower lobe was extremely congested but not consolidated. The pericardium contained 4 oz. of thick pus. The wife, aged 58, was admitted on April 28, nine days after the death of her husband. She said she had been ill in bed for two weeks previously. She was cyanosed, very short of breath, and moribund on admission; there was dullness at both bases with deficient vocal resonance and fremitus; the apex-beat was not visible or palpable, the sounds were very weak, and the cardiac dullness could not be percussed out, as the whole of the front of the chest was dull. She died the next day. At the autopsy there was an empyema at both bases; the heart was covered with thick, purulent lymph, and there was a small

amount of turbid effusion in the pericardium. There was no question of operation in these two cases, even if the pericardial effusion had been diagnosed, which it was not, as the patients were both in a moribund condition on admission. Unfortunately, even in the cases in which a suppurative effusion was rightly diagnosed, and an operation for its evacuation performed, the prognosis was extremely unfavourable, and he had not seen a case of recovery.

He had had, last year, the satisfaction of diagnosing effusion in a case of serous pericarditis, the presence of which was confirmed by operation. On June 1, 1909, he was asked by Mr. Collier to see a case of pericarditis in a woman aged 46, who was under his care in St. Mary's Hospital, for closure of an old colotomy wound. On examining the heart, the apex-beat was palpable in the fifth place in the ventricular vertical nipple line. A friction rub was heard at the apex and in the fourth interspace to the left of the sternum. The area of cardiac dullness extended to the mid-sternal line, and to the left merged into a dull area over the left base, where there was a small pleural effusion. The temperature was 99·6° F., pulse 108, respiration 24. On June 2 the pericardial rub was heard at the base in the second space on both sides of the sternum as well as at the apex. On June 3 the area of cardiac dullness extended $\frac{1}{2}$ in. to the right of the sternum, the friction rub had disappeared from the apex and was only faintly audible at the base. On June 4 the friction rub had entirely disappeared, the apex-beat was not palpable, the heart sounds were distant and faint, and the patient was greatly distressed. Temperature 101° F., pulse 126, respiration 26. The area of cardiac dullness when the patient was lying on her back extended one finger's breadth to the right of the sternum; to the left it merged into the dullness at the left base. On turning the patient on to her right side the area of cardiac dullness to the right of the sternum was *markedly increased*, extending rather more than two fingers' breadth to the right of the sternum. To this point he attached considerable importance, as if the dullness had been due merely to dilatation of the heart it was highly improbable that change of position could influence its area to any appreciable extent, whereas fluid in the pericardium would naturally gravitate to the lowest point. The disappearance of the apex-beat and enfeeblement of the heart sounds also afforded conclusive evidence. He therefore advised operation on the pericardium. Mr. Collier operated, under eucaïne, cutting down in the fifth left space, just to the left of the sternum, and then inserting an aspirating needle. He drew off 30 c.c. of blood-stained clear serum. The patient was greatly relieved by the operation. The

next day a friction rub was audible in the second, third, and fourth interspaces to the left of the sternum, the area of cardiac dullness extended only as far as the sternum, and the apex-beat was just visible and palpable. She subsequently made an uninterrupted recovery, the colotomy wound was successfully closed, and she left the hospital cured and quite well on August 14.

It would appear that the removal of a comparatively small amount of fluid was sufficient to relieve symptoms and promote absorption of the remainder. He thought that effusion was more frequently met with in adults than in children, and that the prognosis was more favourable. Nothing could be more unfavourable than the prognosis in the relapsing pericarditis so frequently seen in rheumatic children, in which effusion was seldom met with, but the pericardium became adherent to the dilated heart.

Dr. W. P. HERRINGHAM said he had only one or two facts which he thought were worth adding to the discussion. It was often said that the heart sank back into the bottom of the sac when there was much fluid. He remembered one case in which there was an effusion to the extent of 24 oz., in which friction over a wide area of the præcordia was audible up to the last. That showed that the heart was right in front at the time, and that it did not always sink back. Another case, the record of which he came upon on looking up the notes, was one in which the pericardium was found after death to be full of air. No connexion with the lung could be found, and it was thought that in that case some gas-forming organism had caused the pericarditis. But no culture could be grown, of either aerobic or anaerobic bacilli, so that if there had been such organism it must have been dead when the patient died. With regard to the causation of the cases in his own wards, they had been pneumococcal, staphylococcal, and streptococcal. He was excluding cases of tuberculous pericarditis, of which there had been in his wards three in the last three years. He remembered one case in which the causation of the pericarditis was rather unusual, in that it was part of a pneumococcal infection which was in the abdomen, not in the lung. He was called to the case by one of his surgical colleagues. He gave it as his opinion that there was pericardial effusion; the surgeon opened the thorax and found it to be so. The patient also had pneumococcal peritonitis, from which she recovered. He had seen two cases in which the dilated heart was punctured, in the belief that the pericardium was full of fluid. In neither case did the puncturing appear to do any harm.

Dr. W. PASTEUR: The few remarks I should like to contribute to this interesting discussion have been prompted by the experience I have gained during the last two years in the bedside examination of patients with a portable modification of the orthodiagraph. A previous speaker expressed the belief that the X-rays would be likely to give us valuable and reliable information in the diagnosis of pericardial effusion. I am fully convinced of this, and would take this opportunity of urging on physicians the importance of acquiring the habit of using the orthodiagraph at the bedside and screening their own patients, not only with reference to pericardial conditions, but in the investigation of morbid conditions within the chest generally. Time will not permit, nor is this the place to explain the method of using the instrument, but I shall be very pleased to demonstrate its use to any one who may be interested in the matter. I should like to say, however, that the orthodiagrams, or shadow tracings, obtained with it represent a distinct advance on the best X-ray photograph obtainable by any other method, if only for the reason that they reproduce the actual size and true relations of the shadows seen on the fluorescent screen.

Before referring to the orthodiagrams I have placed on the screen, I must mention one other point which has been fully elaborated elsewhere:¹ I refer to the effect of arrest of diaphragmatic movement on the base of the lung. Whenever, as the result of paralysis or of reflex inhibition, the action of the diaphragm is suspended for a sufficient time, the lower lobe of one or both lungs is found to become collapsed and airless to such an extent that it sinks entire in water. This condition, which is best studied in cases of diphtheritic respiratory paralysis, is due to the active exercise of the elastic property of the lung, determined by the withdrawal of the expanding force of the diaphragm on the pulmonary bases. Examples of this active collapse—that is, of a lobar collapse occurring in the absence of bronchial obstruction or pleural effusion—have now been observed in connexion with various acute painful affections of the chest and abdomen which have this feature in common—namely, the presence of a powerful source of irritation in the vagus area on either side of the diaphragm. If, therefore, it can be shown that pericardial inflammation inhibits diaphragmatic movement, I submit that this affords another and an adequate explanation of some of the physical signs met with in this condition.

So far I have only had the opportunity of examining two cases of pericardial effusion. The shadow-tracings from these two patients are

¹ Bradshaw Lecture, Royal College of Physicians, 1908.

suspended on the screen. The diagnosis in both rested on clinical grounds, as neither patient underwent operation and both made a complete recovery. In both cases the diaphragm was absolutely immobile, on both sides in the case of a child with a large effusion, on the left side only in the case of an adult with a moderate effusion. As both these patients were examined with especial reference to the movements of the diaphragm, the state of the lung bases was not noted; but this in no way invalidates the point I wish to make—namely, that pericardial inflammation may, and sometimes does, lead to complete arrest of diaphragmatic movement, and that if the inhibition is complete and maintained for a sufficient time, massive collapse of the left base must inevitably follow, giving rise to the usual signs of that condition: i.e. dullness with or without tubular breathing. Further observations will decide whether this inhibition of the diaphragm is a constant or only an occasional phenomenon in pericardial effusion, whether or no it occurs in pericarditis without effusion, and how frequently it is associated with physical signs at the left base. My experience so far leads me to believe that it does not occur in affections of the heart itself. Should further investigations confirm this impression, the sign should prove of considerable diagnostic value.

In conclusion, I would reiterate my conviction that the systematic screening of patients with the portable orthodiagraph is likely to afford reliable information on many points about which our present knowledge is only inferential, and concerning the explanation of which there are still wide differences of opinion.

Dr. J. PORTER PARKINSON: The difficulty of diagnosis of pericarditis is well known, especially of those cases that come on insidiously without local pain and without friction sounds being heard. It is on this account that I wish to mention the occasional presence at the commencement of a pericardial effusion of a sign which is usually supposed to be one of late development only. I refer to signs of compression of the lower lobe of the left lung. I have come across several cases in children during the last few years where this sign preceded by some days both friction and increase of the cardiac dullness. At this period dullness, and weak breath-sounds (sometimes tubular in quality) and fine râles may suggest the onset of pneumonia; but the other signs do not as a rule coincide with this—the temperature is lower and the pulse respiration ratio not altered. In these cases the inflammation of the pericardium probably begins in its posterior part, and there is a fairly considerable exudation before it

reaches the anterior part of the pericardium and a friction sound is audible. One example will be enough to illustrate it :—

J. S., aged 11, was admitted under my care into the Queen's Hospital for Children for chorea of a few days' duration. The boy had had a similar attack five years previously. There was a considerable amount of twitching, and during the first three days the temperature rose to 101° F. without any signs of a complication. Under aspirin this fever disappeared, but for a week he was restless and unmanageable, shouting and screaming, and imagining he heard people speaking to him. At the time of admission systolic and mid-diastolic murmurs were heard at the apex, which was in the line of the nipple. There was no increase of the cardiac dullness upwards or to the right. On December 14 the temperature began to rise, and from this time till January 4 it rose daily to 101° F. or 102° F.; the pulse, too, which had previously been between 90 and 98 a minute, rose to 130. The heart was carefully examined daily, but there was nothing to indicate any fresh lesion. On December 20 I found an impaired percussion note and weak bronchial breathing at the base of the left lung behind, extending upwards to an inch above the angle of the scapula and outwards to nearly the posterior axillary line. As there was no great increase of fever or respiration rate, I suspected this was due to compression of the lung by pericardial effusion, but in front there was no extension of the cardiac dullness and no friction sound. Two days later a friction sound was heard over the base of the heart, and the cardiac dullness had increased upwards and laterally. As the signs of pericardial effusion increased, the dullness at the base of the left lung became more marked and occasionally inspiratory crepitations were heard. All the usual symptoms of pericarditis were well marked. The effusion began to disappear after the temperature fell to normal on January 4, and, *pari passu*, the signs of compression of the lung decreased.

Of course, I am quite aware that these pressure signs are well known : they have been fully described by the late Dr. Sansom. My point is that they may occur before any considerable increase of the cardiac dullness has come on, and so may suggest pericardial effusion at an early stage. If one looks at anatomical plates of horizontal sections of the chest, it is plain how easily a slight collection of pericardial fluid in the case of a patient lying on the back may compress the lower part of the left lung, even before it causes any alteration in size of the pericardial sac as seen from the front.

The second point to which I wish to allude is the question of

paracentesis for the relief of a purulent effusion. I may say I have never found it necessary to remove a non-purulent collection, as they are usually absorbed spontaneously. It is generally recognized that there may be extreme difficulty in diagnosis between pericardial effusion and dilatation of the heart with general pericardial adhesion, and though, as Dr. Herringham has said, puncture of a dilated heart may sometimes do no obvious harm, it certainly does no good. I think, then, that in cases where there can be any doubt as to the diagnosis it is preferable to call in a surgeon who can cut down slowly, feeling his way, and can ascertain the actual condition of things before the pericardium is opened. In a case of mine, a year or two ago, this course was taken, and we found, instead of the expected effusion, a general pericardial adhesion with a greatly dilated heart. I was extremely thankful that I had not plunged a needle into the chest of this patient. No doubt in some skilled hands an exploring syringe may be used with a light heart, but I think for the large majority, if it be necessary to remove pericardial fluid, the method I have mentioned is attended with less danger of harm to the patient.

Dr. CYRIL OGLE: I have collected the post-mortem and clinical records of the last ten years at St. George's Hospital, and Dr. F. W. Higgs has kindly helped me as regards the preceding five years. The main object of the present discussion would seem to be to try to ascertain in what circumstances pericardial effusion should be operated upon and in what way an operation can best be done, either on account of (a) the large amount of an effusion, or (b) its infected purulent character. As regards large amounts of effusion found post mortem, and excluding all those not actually measured or with less than 3 oz. of fluid, the notes from St. George's Hospital show: 16 cases clearly rheumatic in origin (3 oz. to 40 oz.), 13 cases with lobar pneumonia (several of 12 oz. and 20 oz.), 7 cases with pyæmia (either turbid or actual pus), 4 cases with kidney disease as the chief pathological change (two were blood-stained), 2 cases associated with growth (12 oz. and 42 oz., blood-stained), 1 tuberculous, and 13 passive effusions, (15, 17, 20, 42 oz., &c.). There were also a good many cases in infants, associated with pleurisy, to which I refer later on. These were often purulent, also two or three purulent effusions in adults, of obscure origin.

To take the rheumatic cases, sixteen in number. In these the effusion of fluid lay between 3 oz. and 40 oz. (3, 4, 7, 8, 12, 15, 15, 18, 20, 20 and 40 oz.). Apart from fluid, there was often much lymph, the amount of which was not measured, nor can its importance during life,

in clogging the action of the heart, be estimated from post-mortem observations. Indeed, it would not seem possible that post-mortem records can ever indicate the importance or not, during life, of an effusion into the pericardium, as the amount and tenacity of the fibrin, the rapidity or slowness of the whole effusion, must make all the difference in the effect produced. The pericardial sac is an extremely tough and unyielding structure when as yet unsoftened by inflammation. Again, is an effusion, for instance, of 20 oz. associated with a large heart weighing 15 oz. of more importance than one of 10 oz. with a heart weighing 7 oz.? These post-mortem results show, I think, only that when a patient dies with rheumatic pericarditis, there may be found a considerable amount of effusion, and that therefore during life also extensive dullness and other such signs are probably as often due to effusion as to dilated heart, although the two may co-exist.

In turning to the clinical notes corresponding to these cases one finds, as so often is the case, that special points, such as "Rotch's sign," "Bamberger's sign," "pulsus paradoxus," are not mentioned at all in hospital notes, or the physical signs found are recorded so ambiguously as to be valueless in estimating the importance of these special points. There are, however, two examples of "pulsus paradoxus":—

(1) Woman, aged 33 (1233 of 1908): Pericardium greatly thickened and adherent to heart; considerable pockets of pus; a condition also of matting and thickening of mediastinum.

(2) Man, aged 35 (in 1909): Post-pneumonic, purulent pericarditis; not much adhesion to sternum, but dense adhesion to lungs and diaphragm; much lymph and 2 oz. creamy pus in sac, wall of which was $\frac{5}{16}$ in. thick.

In neither of these cases, therefore, was there a large effusion, but rather a condition of thickened and adherent pericardium with matting around, the result of a smouldering purulent infection.

Two instances also of examination by X-rays, in one of which this method seemed to be of help:—

F. C., man, aged 53 (767 of 1902): Præcordial dullness large; heart inaudible; cyanosis. By X-ray screen: "Right half of diaphragm is well visible; there is a dark convex shadow, without movement, bulging to right of sternum. The shadow is continued to the left, well outside the normal position of heart. No cardiac pulsation could be seen as regards the shadow." Operation revealed 115 oz. of serum in pericardium. Recovery.

Rotch's Sign.—I cannot see that true dullness in the fifth right intercostal space (Rotch's sign), even though it be taken as a proof of

fluid in the right inferior pocket of the pericardium, can be of value as an indication that there is a large effusion rather than a large heart plus a little effusion; and it is the relatively large or troublesome effusion which one would wish to be able to be sure of—at any rate, in rheumatic cases and in passive effusions.

Bamberger's Sign is a peculiar one: an area of dullness and tubular breathing about the middle of the left lower lobe at the back, near the spine; at first merely a couple of inches across, but increasing in size, and not due to effusion of pleural fluid, as is shown by the axillary base remaining resonant, but presumably due to collapse of lung. I have fairly often observed it in rheumatic pericarditis, with large præcordial dullness. I have also often tried to test the point, and feel certain that this peculiar sign is not found associated with simply enlarged hearts, such as the large heart of aortic regurgitation or of renal disease. One is also very sceptical as regards the possibility of an enlarged left auricle so pressing on the resistant left bronchus as to produce collapse of the left lower lobe of the lung. I should attach much importance to this sign (Bamberger's) if clearly marked, as indicating a pericardium distended with effusion, although it is difficult to say by what exact mechanism it is produced. [It is possible, I think, that it may be due to compression of the soft descending branch of the left bronchus by the steady pressure of a sac of fluid—an effect which might be thus produced, but might not be produced by the intermittent pressure of a contracting and relaxing enlarged heart.]

The Veins in the Neck.—Whilst observing a patient, a few years ago, who was subsequently operated upon and proved to have the pericardium distended with much fibrin and 16 oz. of pus, I was struck by the condition of the large *veins* in the neck. The external jugulars were permanently distended, even when the patient was sitting up, and showed no pulsation at all; no flicker even. I thought that this immobile distension might be of value, as distinct from the full veins with pulsation which one usually gets with dilated heart, and might indicate an obstruction, by pressure of the fluid, to the entry of blood into the heart, in a case of pericarditis—which is really the point of importance one wishes to arrive at—that is, whether an effusion is exerting injurious pressure. Since then a few cases which I have been able to collect would seem to favour this view. Thus:—

(1) Man, aged 36: Operated upon, and masses of fibrin and 16 oz. of pus found, without mediastinitis. "Veins full and not altering during inspiration; without any pulsation or even flicker."

(2) Man, aged 53 : "Veins in neck full." About five pints of serum were liberated, by operation, from the pericardium.

(3) Man, aged 52 : "Veins in neck full, with no definite pulsation in them." Died subsequently and had 28 oz. of serum in pericardial sac, but heart weighed 21 oz.

(4) Child, aged 1 year and 8 months : "Veins in neck full." Post mortem, pericardium much distended with creamy pus ; layers of lymph also.

It is possible that immobile distension of cervical veins may be of value in conjunction with other signs of pericardial distension, as an indication of actual pressure within the sac. So that, if I obtained a large præcordial dullness, with dullness also extending outwards in the second left space without pulsation there, Bamberger's sign, and distended, immobile, jugular veins, I should feel fairly sure that there was a considerable effusion in the pericardium. Whether one operate or not in such conditions in a rheumatic case is a separate problem, taking into account the natural history of rheumatic pericarditis, with its heart dilatation and effusion, both of which tend to subside, and of which the dilatation is, no doubt, the more important. The urgency of symptoms must, I think, decide.

Perhaps these post-mortem records are of interest in indicating the origin of pericarditis in relation to causation : for instance, its frequent association with lobar pneumonia, and the liability then to become purulent if the patient live ; also of value in showing how frequently in pneumonia it goes undetected. In only three or four out of the thirteen cases was a note made suggesting the presence of pericarditis. [No doubt the sure detection of pericarditis in pneumonia may be extremely difficult, even if kept in mind, on account of pleuritic rub near the heart, adventitious sounds in the lungs, rapidity and distress of breathing, and the like.]

The Association with Pneumococcal Pleurisy.—In 1906 I collected all the cases of pericarditis in children under four years of age who had died in the hospital during the preceding five years. They were sixteen in number ; one was streptococcal and another tuberculous, but the remaining fourteen cases were almost certainly associated with pneumococci. This was proved by bacteriological examination of the exudate in seven of the cases, and in the rest it may be safely assumed from other evidence, such as the frequent co-existence of broncho-pneumonia. All of these cases (save one) were associated with some implication of the pleura—either recent lymph, adhesions, or localized, often inspissated, empyema, which had been treated or not. During the same period

there were twenty-three cases of fatal broncho-pneumonia in infants under four years of age, in which the pleuræ were normal, as expressly stated in the post-mortem records. In none of these twenty-three cases was there any pericarditis.

These facts would suggest that pneumococcal pericarditis in infants arises only by extension of infection from the adjacent pleural cavities, and not, as may inflammation in a joint, by general pneumococcal blood-infection. Of these fourteen cases of pericarditis in infants, there were five which were examples of manifest pus in the pericardium, and four others which may be so considered, but in an incipient stage; turbid serum infected with pneumococci. During life it would seem that the existence of pericarditis had escaped detection in all of these cases; indeed, in only one case out of the sixteen was the possibility apparently discussed; so that the post-mortem results were, in all, nearly entirely unexpected. Nevertheless, on reading these records one would say that in some of the cases, at any rate, the state of the pericardium had much to do with the death of the child, as the other conditions present, such as a drained empyema, would seem to be of minor importance.

I think that there are, at any rate, two causes why pneumococcal pericarditis escapes detection: first, in connexion with the sign of friction; and, secondly, that it is not suspected and looked for in certain conditions in which it is apt to arise. It is well known, as regards friction, although it is not perhaps sufficiently emphasized, that purulent pericarditis gives no friction sound; so that, if the presence of friction be relied upon as a necessary sign in pericarditis, then pericarditis will not be suspected. But when the material called "pus" is present in the pericardium, there is always, as far as I have observed, lymph also, as a solid, sticky exudate in addition, and it can hardly be doubted but that, in an earlier stage of the inflammation, friction is always present; it may be that it could have been heard for only a short time.

One is led, therefore, to dwell on the great importance of careful and repeated examination of the pericardium, in order to detect a transient friction, in all cases of pneumonia in the adult and in pleural implication in a child; as otherwise, if the friction be overlooked, an important clue may have been missed as to the actual condition existing, when perhaps an enlarged præcordial dullness with muffling of sounds and absence of heart-beat is subsequently found.

The following is a tabulated record of the operations upon the pericardium during the period under review.

OPERATION BY NEEDLING.

Girl (5) 1712 of 1900	Rheumatic	Needle into fifth right space; 4½ oz. serum withdrawn; this done after unsuccessful excision of fourth left cartilage	Died same day	Recent lymph serum, 4 oz. heart, 11 oz., with fine vegetations
Girl (11) 1791 of 1901	Rheumatic	Needle—unsuccessful—when moribund	Died in half an hour	Recent lymph; serum, 3 oz.; heart, 12 oz.; vegetations on all valves
Man (58) 414 of 1903	Lobar pneumonia; granulated kidney	Needle into pericardium by mistake, through pleura, in tapping pleural cavity	Died four days afterwards	Recent adhesions; pure blood in sac, 3½ oz.; wound in left ventricle
Boy (14) 1326 of 1905	Rheumatic	Needle; 1 dr. blood-stained serum removed	Recovered and discharged	—
Man (19) 1129 of 1908	Rheumatic	When almost moribund: (a) needle in sixth left space, which was clogged with fibrin; (b) needle in epigastric angle, which did not enter the pericardium	Died soon afterwards	Much fibrin; serum, 40 oz.; heart, 24 oz.; lightly adherent in front; valve mischief

OPERATION BY REMOVAL OF FOURTH OR FIFTH LEFT CARTILAGE.

Girl (5) 1712 of 1900	Rheumatic	Piece of fourth cartilage removed; pericardium supposed to be opened, but was not; subsequent removal of fluid by needle in fifth right space	Died same day	Post-mortem results as shown above
Man (24) 1010 of 1900	Rheumatic	Piece of fifth left cartilage removed; left pleural cavity opened; 1 oz. serum removed and drainage of 1 oz. more from pericardium	Recovered and discharged	—
Man (36) in 1899	Purulent pericarditis; inspissated pus near pericardium	Piece of fifth left cartilage removed; fibrin and 16 oz. pus under pressure removed	Died fourteen hours afterwards	Much fibrin still; ante-mortem clotting in right branch of pulmonary artery; valves healthy

As regards operation. I suppose that in the acute phase of lobar pneumonia no operation would be contemplated by anyone. A patient in this condition, with pericarditis, almost always dies, and would probably die the sooner if operated upon. But it is otherwise in the few cases where the infective pericarditis subsists almost alone, after the acute symptoms of pneumonia, for instance after the crisis, as does an empyema. So also in some cases in children, as I have above mentioned,

where apparently the collection may have existed for a considerable length of time. And, again, in those cases in purulent pericarditis in adults, which appear to be "primary," but which, as I believe, have most probably arisen by extension of pneumococcal infection from old pleurisy adjacent to the heart. For these, not mere puncture, but pericardotomy and removal of masses of infected lymph and drainage would seem indicated. In this relation, may I allude to a mode of operation by incision in the epigastrium, which I published in 1900, in conjunction with the late Mr. Herbert Allingham. It was suggested by difficulties encountered in an operation on a case of purulent pericarditis where 16 oz. of pus were present and a great amount of fibrin. This operation was done in the ordinary position, by removal of some of the

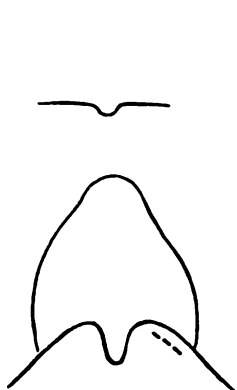


Diagram showing seventh costal cartilage, pericardium, and line of incision.

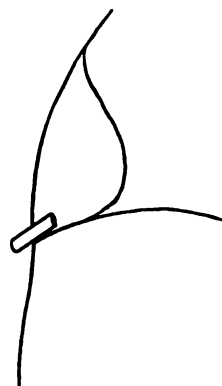


Diagram showing pericardium, diaphragm, and drainage-tube in position.

left fourth cartilage. One, however, wished for some opening in a more dependent position, and I worked out, in the post-mortem room, the details of a possible operation by incision in the epigastrium and found that it was quite feasible, and in children especially, even without removal of any cartilage at all. The steps of this operation by incision along the lower border of the seventh left costal cartilage, in the epigastrium, and then through the costal attachment of the diaphragm (*see diagram*) are fully described in the *Lancet*.¹ The advantages are that:—

(1) The left pleura cannot be injured, as it is far away in the normal arrangement of the organs and would be still further removed in pericardial distension.

¹ *Lancet*, 1900, i, p. 693.

(2) Drainage is from the most dependent part of the sac, when patient is half propped up, and is through a large opening not bounded by cartilage or sternum.

(3) Great ease is afforded for the exploration and cleansing of the heart, back and front, to its extreme limits.

Since this method was published, we have had five examples of the operation at St. George's Hospital; the details of these are annexed:—

FIVE CASES OF PERICARDOTOMY BY EPIGASTRIC INCISION.

1	Man (53) 1019 of 1902	Hydropericardium	115 oz. serum containing cholesterin	Recovery, and able to do labourer's work; seen six years afterwards	Dr. Latham and Mr. Pendlebury
2	Boy (12) 1005 of 1902	Rheumatic pericarditis	Serum under pressure let out; drainage; wound soon healed	Died six days afterwards from cardiac disease	Mr. Pendlebury
3	Man (31) May, 1904	Lobar pneumonia in late stage	4 oz. serum and fibrin removed; drainage; wound healed in fourteen days	Recovery; well at present time, six years after operation	Dr. Penrose, Dr. Ogle, and Mr. Pendlebury
4	Boy (3½) August, 1902	Exploration of pericardium, on account of pneumococcal empyema near heart	Pericardium found healthy; wound sewn up; prompt healing	Lived a month longer and then died of tubercle; pericardium then found normal	Dr. Penrose and Mr. Allingham
5	Woman (53) 1686 of 1902	Tuberculous pericarditis	10 oz. serum and fibrin removed; drainage; wound healed well	Lived for five months afterwards; died with generalized tubercle	Dr. Ewart and Mr. Allingham

I think that these five cases show, at any rate, that the operation itself can be a very satisfactory one, as regards the objects aimed at; thorough exploration with the finger, and cleansing of the sac and heart, back and front, from infected material, which may often be in pockets, and then uninterrupted drainage from the most dependent part and rapid healing.

I am not at all sure but that incision down to the pericardium and removal of clogging masses of fibrin, as well as the fluid, in even the serofibrinous effusion of rheumatic pericarditis, whenever, though *rarely*, it is decided to operate at all, is not a better plan than puncture with a sharp needle, in uncertainty as to where the needle penetrates and with the probability of its being clogged with fibrin and of the necessity of repetition of the process. If pericardotomy be decided upon in a case, I think that the epigastric method will offer many advantages over

operation in the more usual positions. At any rate, the records which I have been able to trace, and which are mentioned above, of needling and of anterior pericardotomy, do not seem satisfactory in their immediate results for one reason or another.

Dr. IRONSIDE BRUCE said that he had had the opportunity of demonstrating the presence of pus in the pericardium by X-ray in two cases. The first one was a case of acute osteomyelitis, in which, clinically, pus in the pericardium was suspected. On X-ray examination its presence was clearly to be made out. He exhibited an enlargement of the radiogram of this case and, for purposes of comparison, a similar enlargement of a normal thorax. He thought the contrast was sufficiently obvious,

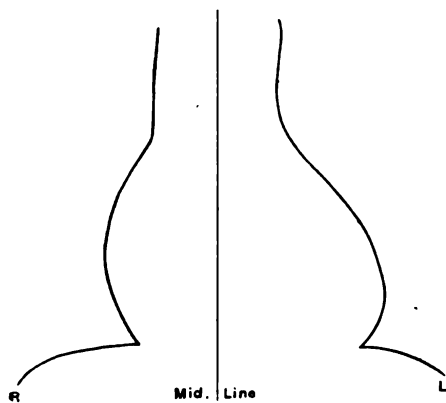


FIG. 1.

Normal cardiac opacity.

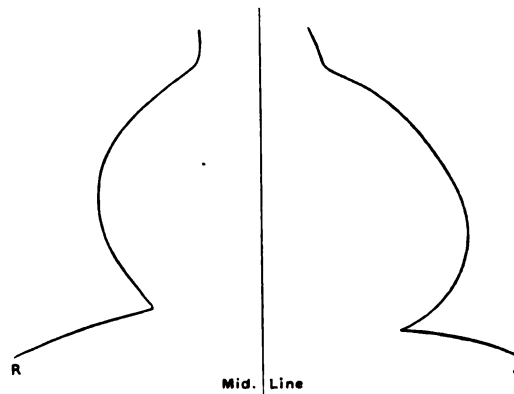


FIG. 2.

Cardiac opacity in pericardial effusion.

the cardiac capacity showing a very definite difference in size and shape in the two cases (figs. 1, 2). When pus was present the cardiac capacity was spherical in shape and its outline sharp, whereas the normal cardiac opacity was not in any way spherical in shape, and its outline, owing to the movements of the heart during the exposure, was blurred. In this case there was no doubt as to the presence of pus, for the pericardium was opened by Mr. Tyrrell Gray, and pus evacuated. The second case was one which had already been reported to the Section¹. The radiogram shows the same appearance of the cardiac opacity, but in this case the presence of an evacuated empyema of the left pleural cavity

¹ *Proc. Roy. Soc. Med.*, 1908 (Clin. Sect.), i, p. 15.

displaced the cardiac opacity towards the left, otherwise the spherical shape of the pericardium containing pus can be observed, as also sharpness of outline. Dr. Bruce expressed the opinion that on X-ray examination the appearance presented by the cardiac opacity is sufficiently characteristic when pericardial effusion is present to make the diagnosis of this condition a matter of no great difficulty.

Mr. Lyster said, in response to the President's invitation, that he had little to communicate, as he had come to learn. He had not had much opportunity of examining pericarditis, but it seemed clear that the definite outline and the spherical shape of the pericardium seen by X-ray examination formed the most marked features. The ordinary movement of the heart could not be seen, especially low down, when there was effusion. He thought he had detected, in one case, that there was some movement at the upper part of the pericardium. But at a previous examination there was no movement of the heart visible at all. He had no doubt it was possible by means of the X-rays to make a distinct diagnosis of even small quantities of fluid within the pericardium.

Dr. F. J. Poynton said his remarks would be confined to the condition in children; and he had to thank Dr. Owen, of University College Hospital, and Dr. Frew, of Great Ormond Street Children's Hospital, for helping him with the elaborate records which they had prepared. A large pericardial effusion in a child was an exceptional event. Though he was interested in rheumatism, he had not seen a case of rheumatic pericarditis in a child which had needed an operation on account of the amount of effusion. In 1907, in a discussion at the Medical Society of London, he pointed out that pneumococcal pericarditis in children was a disease, to a great extent, of very young children. He there analysed the records of 100 cases, and found that 66 per cent. of them were under 3 years of age, and 80 per cent. under 4 years. He was not in agreement with those who looked upon that pneumococcal pericarditis as spreading from the lung or pleura; the evidence, in his opinion, was not at all in favour of that. He thought it was frequently a direct infection. One of the most interesting points connected with that form of pneumococcal pericarditis was the length of time that it lasted. The child might have pericarditis for months, judging from the history and the post-mortem details. There seemed to be two main points in the present discussion. The first was as to how often a pericardial effusion was, by its mechanical properties, a danger to the

patient; and the second was how often one ought to drain the pericardium to get rid of the noxious fluid. In pneumococcal pericarditis one should, theoretically, always drain the pericardium if the condition was diagnosed. But when one studied the condition of pneumococcal pericarditis as it showed itself, one found that 15 per cent. of the cases met with had suppurative meningitis, and in 10 per cent. there was suppurative peritonitis. So his own attitude in regard to a doubtful case of pneumococcal pericarditis in an infant was one of great caution; and he did not believe in being in a hurry to explore, because if one were wrong one might kill a patient who was already suffering from a double or single empyema or pneumonia. Over 80 per cent. of those cases in children were associated with either pneumonia or empyema. He did not see much hope for surgery in pericarditis; the best chance seemed to be in the future by serum treatment. He wished, in conclusion, to refer to a branch of investigation which had not received sufficient attention in the discussion—namely, that by experimental investigation. He thought it was a remarkable fact that from the disease which was the greatest cause of heart disease—namely, rheumatism—it was possible to get from the pericardial effusion an organism by which one had proved many of the points now under discussion. One could, in an animal, cause death from a dilated heart without pericarditis, produce pericardial effusion, and show the relations of endocarditis, simple and malignant. Experiment would enable us to stand on much safer ground with regard to the pathology: for example, that dilatation of the heart was not the result of spread of inflammation from an inflamed pericardium, in that it might occur without pericarditis, as emphasized by Dr. Lees, was verified by experiment.

Dr. Box: Primary pericarditis appears to be as rare as is a primary pleurisy. Of the 112 cases collected from the records of St. Thomas's Hospital, pericarditis occurred as an isolated lesion in only two, each instance being in a chronic alcoholic. With these exceptions, the nearest approach to a primary condition is where inflammation simultaneously involves two or more of the great serous sacs without evidence of implication of the viscera; but cases even of this sort are exceptional, and only three or four are recorded among our notes. The combined serous effusions may be simple or purulent, and are mostly pleural and pericardial; but in one instance (suppurative) the peritoneum was involved as well. As a general rule, pericarditis arises in combination with other disease, and accompanies such conditions as acute rheumatism.

with endocarditis (the latter lesion often being of old standing), pleuro-pneumonia, broncho-pneumonia, various forms of pyæmia and chronic renal disease. In our series the rheumatic, pneumonic, and pyæmic cases are approximately equal, and together account for a little more than half of the total number. Another form which is sufficiently common for special mention is the terminal pericarditis of granular kidney. In children aged under 5 broncho-pneumonia, either discrete or confluent, and sometimes accompanied by empyema, takes first place as a cause, but is run very closely by pyæmic conditions, mostly dependent upon acute bone disease. The latency of pericarditis is exemplified in a striking manner; no fewer than 51 of the 112 were unsuspected during life (over 40 per cent.). The latent cases were mostly pyæmic, pneumonic, broncho-pneumonic, or associated with empyema.

SOME CHARACTERISTICS OF THE VARIOUS FORMS OF PERICARDITIS.

The Rheumatic Cases are those in which the most explicit accounts of increase in the area of præcordial dullness are forthcoming. The dull area is largest, as a general rule, in cases of this class, but its mere size affords no indication of the amount of effusion present, being in great part due to the actual size of the heart itself. Most of the rheumatic cases are complicated by old endocarditis; in addition the heart is dilated, and recent vegetations are often present on the mitral and aortic valves. The effusion is serous, sero-fibrinous, or blood-stained; in one case only was the exudate composed of "flaky pus."

The Pneumonic Cases.—Where pericarditis complicated pneumonia, death, as a rule, occurred at the height of the disease, and the pericardial inflammation was only discovered at the post-mortem examination. In three instances the course of the fever was prolonged for three weeks, and empyema was consequently suspected. In one of these cases there was no empyema, but an unsuspected pericarditis. In the other two the empyemata were found and drained, but the pericarditis again overlooked. Two cases appear to conform to the type on which Dr. Samuel West has laid stress. In each the temperature was normal at the time of death and pericarditis entirely unsuspected. The interval since the onset of the pneumonia was fifteen days in one instance and longer in the other. In all, save one, of the cases attributed to pneumonia the pericardial effusion was sero-purulent or purulent. The hearts were not so greatly or so invariably enlarged as in the rheumatic form of the disease. Valve lesions were absent in all save one. The two heaviest

hearts were taken from patients in whom a mixed nephritis was also present and weighed $13\frac{1}{2}$ oz. and $14\frac{1}{2}$ oz.

The Renal Form.—This is essentially the pericarditis of granular kidney. The onset is insidious, præcordial pain often absent, and the temperature usually normal or subnormal; even in the two cases which were exceptional in the last respect the temperature only rose at the time of death, some days after the discovery of the pericardial friction sound. The pulse-rate is not necessarily increased on the supervention of the pericardial inflammation. Vomiting, which is possibly uræmic, may occur. The enlargement of the area of præcordial dullness is due rather to cardiac hypertrophy than to pericardial effusion. Friction was well marked and formed the main diagnostic feature in all our cases. In most instances the pericardial fluid was scanty and serous or turbid. In three cases the effusion was blood-stained and in one purulent. The prognosis is, as a general rule, fatal. The St. Thomas's Hospital reports show that during a period of thirteen years twenty in-patients with chronic renal disease of the interstitial form developed pericarditis, and of these only two survived. Taking all the fatal cases of chronic interstitial nephritis admitted during the same period, recent pericardial inflammation was found in a fraction over 10 per cent.

The Pyæmic Form.—Pyæmic pericarditis was mostly latent. The heart was invariably almost or quite normal in size and was free from endocarditis save in two instances, where recent ulceration was found just above the tricuspid valve. In three instances abscesses were found in the heart wall, and in one of these cases rupture of the wall of the left ventricle had taken place. The pericardial effusion was either turbid or purulent.

The Tuberculous Form.—This must be uncommon. Three cases were found, but have been excluded because they were adhesive and unaccompanied by effusion.

CLINICAL INDICATIONS OF THE NATURE OF THE FLUID PRESENT IN THE PERICARDIAL SAC.

Temperature is no guide. Fever may be as pronounced where the effusion is serous as where it is purulent. Pus *may* be present with a temperature which is normal or but slightly febrile. Neither is the size of the effusion a safe criterion; although serous effusions are, as a rule, larger than those which consist of pus, yet purulent effusions do on occasions equal the largest serous ones. Moreover, all accounts show

how extremely difficult it is to gauge the amount of fluid in a case of pericarditis.

The nature of the accompanying pleural effusion is often a good index to the contents of the pericardium, but this test is not infallible, since pus in the pleura may be accompanied by serum in the pericardium, an event, however, which is decidedly uncommon. In only one instance was the association of a purulent pericarditis with a serous pleurisy found. The nature of the illness which the pericarditis complicates is all important as an index to the contents of the sac. As a general rule, the effusion in the rheumatic cases is serous, in the pneumonic or broncho-pneumonic sero-purulent or purulent, in the pyæmic, turbid or purulent. Considering the circumstances under which pericarditis arises, leucocytosis is likely to be of but little value. It is remarkable that purulent pericarditis was undiscovered in every instance of its presence, save one.

The Significance of a Blood-stained Effusion.—It is stated that hæmorrhagic pericardial effusions are associated with tuberculous or cancerous pericarditis, scorbutic conditions, and pericarditis of the aged. These limitations are not borne out by the cases we have collected. Blood-stained effusion was found in fourteen patients. Exactly half of these were cases where pericarditis was associated with recent endocarditis in patients free from tubercle or malignant disease, and clinically supposed to be rheumatic. Of the rest, one was a case of empyema, one of malignant endocarditis, three of chronic renal disease, one septicæmic, and one a chronic alcoholic. Almost pure blood was found in one case, the wall of the left ventricle having ruptured at the site of a pyæmic abscess. The pericardium was acutely inflamed.

SOME REMARKS ON THE PHYSICAL SIGNS OF PERICARDIAL EFFUSION.

(1) *Upward Extension of the Præcordial Dullness.*—In seven of nineteen cases in which the dull area reached the second left costal cartilage this dullness was proved to be entirely cardiac, whilst in the cases in which it reached only to the third left cartilage the amount of fluid was insignificant.

(2) *Rotch's Sign.*—The descriptions given by English authorities as to the character and mode of experimental production of Rotch's sign do not appear to tally with the account given by Rotch himself. Rotch states that he *did* inject his bodies in the position of orthopnoea,

the trunk being bent on the lower limbs at an angle of 120° , so presumably the sign should be sought for in this posture. In describing his sign he says that it consists of dullness in the fifth right intercostal space, but is careful to state that the criterion which differentiates pericardial effusion from an enlarged heart is "absolute dullness in the fifth right interspace, 3 cm. or 4 cm. ($1\frac{1}{8}$ in. or $1\frac{1}{2}$ in.) from the right parasternal line," evidently using the latter term to denote the right edge of the sternum. The notes at our disposal afford no evidence of the value of this sign.

(3) *The Posterior Signs.*—Signs have been described near the inferior angle of the scapula and below the posterior lung bases. The frequency with which pleural effusions or pneumonic consolidation accompany pericardial effusions seriously detracts from the value of those posterior signs which are held to indicate pericardial distension. Moreover, similar signs, near the angle of the scapula at all events, can be produced by a dilated and hypertrophied heart. Should there be any considerable area of non-vibratile dullness at the posterior lung base, even though tubular breathing be audible at its upper part, near the inferior scapular angle, experience teaches that it is advisable to explore the pleura with an aspirator before concluding that the signs are solely due to pericardial effusion. Of all our cases, only three showed the posterior scapular sign in its typical form—i.e., a circumscribed area of dullness with tubular breathing at the lower scapular angle. In each of these there was massive collapse of the left lower lobe, unaccompanied by any pleural effusion. In one instance similar signs were found in front over the middle lobe of the right lung near the sternum, and here again the middle lobe was found post mortem to be largely collapsed.

THE QUESTION OF OPERATIVE INTERFERENCE.

The field for successful operative measures, whether by aspiration or incision, in pericarditis appears to be small. Where there is any reasonable certainty that a large serous effusion is itself seriously embarrassing the heart, the proper procedure, no doubt, is to remove the fluid; but reasonable certainty as to the size of the effusion is not easy to attain. In the three cases operated upon at St. Thomas's Hospital the amount of fluid withdrawn measured under 5 oz., $4\frac{1}{2}$ oz., and barely 2 oz., the large area of dullness in each case being, as usual, in the main due to cardiac enlargement. Could any means of preventing internal pericardiac adhesions be devised the scope of the operation

would be greatly extended. With this end in view, sterilized liquid paraffin was injected into the sac of one of these patients (Dr. Box and Mr. Corner's case). Purulent pericarditis after empyema, or residual pericardial abscess following pneumonia, should be ideal cases for surgical treatment. Pyæmic cases are, as a general rule, hopeless for other reasons by the time pericarditis develops.

Dr. ALEXANDER MORISON: In an examination of forty-four cases of acute pericarditis and adherent pericardium occurring in the practice of my colleagues and myself at the Paddington Green Children's Hospital, when I was attached to that institution, I found that in the twelve cases in which there was no concurrent evidence of endocarditis as denoted by valvular disease, the acute pericarditis, which in the majority of cases was purulent, was due to extension of the morbid process in tuberculosis, pneumonia, and empyema. The mere absence of concurrent valvular disease has therefore a certain value in distinguishing the pericarditis of local extension from that of a more general or blood infection as one finds in rheumatic fever. The criterion, of course, is not absolute, as pneumococcal endocarditis occasionally occurs. Among names which should be mentioned in this discussion is that of the late Dr. Octavius Sturges, who attached much importance to the percussion signs of variation in the amount of fluid within the sac, as noted at the left cardiac base. This practically corresponds with the position of Sibson's notch, and is the point at which increase and diminution in the contents of the sac may be most easily noted, especially in children. Dr. Sturges gave the results of his ripe experience in the Lumleian Lectures for 1894. Dr. West has indicated that the most important point in diagnosis is to differentiate between a dilated and usually hypertrophied heart and pericardial effusion when it is considerable in amount. He has shown that the muffling of the heart sounds may be marked when only dilatation is present; that pericardial friction may persist in portions of the area of cardiac dullness when accumulation within the sac is in considerable degree; that the pulsus paradoxus is attributable to other causes than pericarditis; and states as the most characteristic sign, in his opinion, the extension of the dullness beyond the apex-beat; although the apex need not, of course, be the actual heart apex. On all these points I quite agree with him, except in so far as he regards the pulsus paradoxus to be due to any other cause than the effect of exaggerated respiration upon the circulation. In agreement with his conclusion as to the most characteristic sign—namely, the extension of the dullness beyond this

virtual apex-beat—I wish to make some remarks upon a sign which has not hitherto been described, for sufficient reasons.

On August 12, 1908, my colleague, Mr. Stabb, at the Great Northern Central Hospital removed from a patient of mine portions of the fourth, fifth, and sixth ribs to accommodate with more space a greatly enlarged and hypertrophied mitral heart. Greater difficulty was experienced in detaching the pleura from the ribs in this case than in one Mr. Stabb previously operated upon, and which is still, two years after the event, deriving benefit from the operation. The patient, who was much distressed by a large pneumothorax for a few days, however recovered from this, and promised to make a good recovery so late as the beginning of October, 1908, when he developed signs of pericarditis—namely, pericardial friction. Fluid accumulated within the sac, and, on coughing, bulged the sac and the attached soft præcordial parietes. Friction could still be heard in portions of the præcordial area, and the amount of fluid it was difficult to calculate. But a cardiographic tracing showed signs which I had not observed before, and which I could not at the moment quite explain, but the explanation of which was, to my mind, made clear by an observation upon another case to which I shall refer.

While the cardiogram of the *sternal* end of the decostated area remained much as before the effusion into the sac, that at the outer part of the sac, beyond the apex-beat as Dr. West puts it, revealed a series of small undulations due to the transmission through the fluid to the recording instrument of vibrations caused by the action of the heart. That is to say, the character of the cardiogram was altered by the intervention of fluid.

The intervention of air has the same effect. In a case on which an Estlander's operation was performed on a patient of mine by Mr. Stabb, a cavity intervened between the heart and parietes which could hold a pint of fluid, which was ejected *per saltum* by the movements of the organ. When the cavity was empty I had attached to the opening in the parietes an indiarubber covering with a tube outlet, to which I attached a Mackenzie's polygraph, and obtained the tracing which I show you, in which you will observe a series of small undulations crowning the respiratory curve, and which are the transmitted cardiac pulsations.

I think, therefore, that the change in the character of the cardiogram of pericardial effusion, on the lines I have indicated, may be of service in distinguishing between enlargement due to dilatation of the heart and that caused by a distended pericardial sac, although the change

will not be so marked in cases which have not been submitted to præcordial thoracostomy. I show you tracings from the cardiac case I have described, and also a photograph of the patient after his operation wound had healed, as well as a transverse section of his heart and pericardial sac obtained after his death, which occurred on October 29, that is, seventy-seven days after the operation. The sac was not opened at once, but was placed in formalin solution for fixation.

On the question of the *treatment* of non-suppurative pericarditis, I would recall the wisdom of a late eminent physician who added to the confusion of one who had failed to detect a pericarditis by remarking that it was perhaps fortunate he had not done so, as he might have been tempted to treat the condition. At an early stage of pericarditis, if the condition be associated with pain, leeching and warmth to the præcordia may promote comfort; but vigorous attempts to subdue inflammation by counter-irritation or cold I regard as equally objectionable. But when evacuation of the sac is contemplated, either for serous or for purulent effusion, I confess that I have a preference for cutting down upon, rather than perforating, the sac. There is admittedly danger in advancing without scouting. In the case of a man under my care at the Great Northern Hospital with greatly extended cardiac area and muffled heart sounds, and with evidences of cardiac failure, I was uncertain whether I had to deal with a dilated heart or with pericardial effusion, and asked my colleague, Mr. Stabb, to remove a portion of the fifth-rib cartilage to determine the point by exploration. This was done, and we found we had to deal with an adherent pericardium. Nothing further was attempted, but with continued rest, and under the influence of cardiac tonics, the patient left hospital considerably improved in health. His progress after the exploratory operation was so favourable that one was tempted to regard it as having been beneficial; but as no more than incision was attempted, it was difficult to explain any association between the operation and his convalescence. To have explored by puncture would certainly, in this case, have been disastrous.

Dr. WEST, in replying on the discussion, said the speeches had left very little for him to add. The debate had been interesting to him in many ways, and especially Dr. Pasteur's reference to the orthodiagraph, and his explanations of the collapse of the lower lobes. Dr. Ogle's was also a very interesting contribution: that gentleman had had an unusual experience in having had such large amounts to remove from the pericardium. Though equally large amounts had

been recorded, they were very rare. Dr. Ironside Bruce's demonstration of the absence of movement of the heart sac where there was effusion had been very interesting, because that was a point on which he (Dr. West) had made some remarks as distinguishing pericardial effusion from dilatation. It was odd that, with large effusions in the pericardium, there should be no pulsation, and though the X-rays showed it, they did not explain it.

Of course, there were many interesting conditions which were more or less excluded by the terms of the discussion. Dr. Herringham referred to a case of pneumopericardium, and he (Dr. West) could have mentioned a case or two of a similar kind, but he had purposely omitted them. He had never previously heard of a gas-producing bacterium in the pericardium, though such might of course occur. He was extremely interested to hear what Dr. Box said; and the contribution was altogether a very valuable one. Dr. Box's experience coincided with his own, that primary pericarditis was very rare. Dr. Box was the only speaker who mentioned granular kidney. He (Dr. West) had not seen a case of hæmorrhagic pericarditis in granular kidney, although such cases had been described. He drew a distinction between effusions which had very little blood and those which had a great deal; and he had never seen anything beyond a mere trace of blood in those cases. Dry pericarditis, or at any rate with very little effusion, was very common, and, as Dr. Box said, invariably fatal.

He had nothing but gratification to express concerning the valuable results which had been recorded by many of the speakers; and he did not doubt that when their statistics were published it would be seen that there was a great deal more in them than the time which had necessarily been allotted to each speaker had made it possible to adequately bring out. He thanked all those who had listened to him with so much patience.

*STATISTICS OF PERICARDITIS WITH EFFUSION, FROM
THE LONDON HOSPITALS, PROVIDED TO FORM A
BASIS FOR THE ABOVE DISCUSSION.*

St. Thomas's Hospital.

By CHARLES R. BOX, M.D., and G. G. BUTLER, M.B.

*A Review of 112 cases admitted between June 30, 1894, and
June 30, 1909.*

DURING the period under review, 112 cases of pericarditis with proved effusion were admitted to St. Thomas's Hospital. This does not represent the total number of cases with effusion admitted during that period, since all cases in which the effusion was not determined by operation or by post-mortem examination have been excluded. The cases were distributed as follows :—

1894 ... 4 cases	1899 ... 5 cases	1904 ... 4 cases
1895 ... 7 ..	1900 ... 6 ..	1905 ... 11 ..
1896 ... 11 ..	1901 ... 4 ..	1906 ... 14 ..
1897 ... 4 ..	1902 ... 9 ..	1907 ... 14 ..
1898 ... 4 ..	1903 ... 9 ..	1908 ... 6 ..

AGE INCIDENCE.

		Males	Females	Total
Under 1 year	...	4	2	6
1 to 5 years	...	9	3	12
5 " 10 "	...	8	4	12
10 " 15 "	...	8	8	16
15 " 20 "	...	6	3	9
20 " 25 "	...	7	3	10
25 " 30 "	...	6	4	10
30 " 35 "	...	4	2	6
35 " 40 "	...	6	1	7
40 " 45 "	...	7	0	7
45 " 50 "	...	8	2	10
50 " 55 "	...	3	2	5
55 " 60 "	...	2	0	2
Over 60 "	...	0	0	0
Total	...	78	34	112

Sex.—Of the 112 patients, 78 were males and 34 females.

Mortality.—In every instance but two the result was fatal. The large proportion of fatal cases is the consequence of the limitations of the investigation.

Occupations.—This varied greatly. Where a terminal pericarditis complicated renal disease, the patients were mostly in the building trade (bricklayer, carpenter, painter, builder's handy-man, labourer, "engineer"): one was a scene-shifter. Among the rheumatic cases were two boys, one a lamp-washer and the other a basket-washer. Most of the patients had occupations necessitating an outdoor life, but this is not remarkable among the class from which the hospital draws its cases.

Previous Illnesses.—Among the 23 patients in whom pericarditis complicated acute pneumonia, 3 had already suffered with lung inflammation, 4 had previously suffered from acute rheumatism, 1 from tonsillitis, and 2 were alcoholics. Of 29 cases supposed to be rheumatic in origin (because they arose out of articular rheumatism, or were complicated by chronic endocarditis), there was a history of previous attack or attacks of acute rheumatism in half. Endocarditis, old or recent, was present in all. In 18 cases acute articular rheumatism accompanied the onset of the pericarditis.

Concurrent Illness.—Acute rheumatism was present in 18 cases, chronic endocarditis in 11 cases. Acute pneumonia was present in 23 cases, complicated by empyema in 5. Broncho-pneumonia was present in 8 cases, all infants or young children. Simple pleurisy accompanied pericarditis in 2 cases. Empyema was present in 18 cases (including 5 cases of pneumonia and 2 of broncho-pneumonia, mentioned above). Empyema was bilateral in 4, left-sided in 9, and right-sided in 5. In one case with bilateral empyema, purulent peritonitis was also present. Malignant endocarditis was present in 2 cases. Pyæmia was present in 18 cases. In 5 this was attributed to infections of wounds, sores, or abrasions, in 1 it followed childbirth, in 1 an abscess of the iliacus muscle, and in 2 was unexplained. The remaining 8 were bone cases. Renal disease was complicated by pericarditis in 14 instances, 12 were cases of granular kidney, 1 of cystic kidney, and 1 of recent nephritis with renal thrombosis. Malignant disease in the mediastinum caused pericarditis in 2 instances. The growth was lymphosarcomatous in one and carcinomatous in the other. In 1 case a chronic abscess of the ovary was associated with pericarditis, of which it was regarded as the cause. Pulmonary phthisis, with lardaceous disease, was found in one case, and another occurred in a patient with chronic tuberculosis of the hip, but in neither of these did the pericarditis appear to be tuberculous. Three cases of tuberculous pericarditis, two in patients with phthisis and one in a child who died of tuberculous meningitis, have been excluded, because they all were adhesive and unaccompanied by effusion.

Course and Duration.—The pericardial inflammation was acute and latent (undiscovered) in no fewer than 51 of the 112 cases (including simple pneumonia 15, empyema complicating pneumonia or broncho-pneumonia 6, empyema alone 3, pyæmia 13, and renal disease 4, with others). Of the cases where pericarditis was recognised during life, the average duration of the disease appeared to be a little over a week in fatal cases. The longest period before death was twenty-eight days, and the shortest less than twenty-four hours.

Symptoms and Physical Signs.—The aspect of the patient is for the most part not described in the notes. Where mention is made of it, it is variously spoken of as "anxious," "distressed," "restless," "flushed," &c. Posture again nearly always escaped record. In only 10 instances is orthopnoea definitely mentioned. In 7 cases the patient was described as recumbent. The presence in most cases of such diseases as valvular lesions and pneumonia renders the notes of posture of little value with regard to the effect produced by pericardial effusion itself. Cyanosis is definitely mentioned in 26 cases, and in all save 3 of these either mitral disease or pneumonia was present. The exceptions were an alcoholic, a patient suffering from advanced phthisis, and one case of granular kidney. Dyspnoea is mentioned in 67 of the 112 cases. In 10 of these orthopnoea was present, and in 10 others the dyspnoea is described as "great" or "well marked." In all the cases of urgent dyspnoea except one, either valve lesions or pulmonary consolidation were found. In the remaining case empyemata were present on each side. Cough was definitely mentioned in 42 cases, but no importance can be attached to the number, since it was frequently not recorded in cases of pneumonia. Whooping-cough was present in one case. Vomiting was only occasionally mentioned as a feature. It was well marked in the case of whooping-cough, and in 4 of the renal cases. It was also noted in 7 of the heart cases and in two of the pyæmic ones. Præcordial tenderness was only mentioned in 4 cases.

The Pain of Pericarditis.—Præcordial pain was present in 19 cases. Epigastric pain was present in 6 cases. Combined præcordial and epigastric pain in 3. Anginoid pain, extending down the left arm, was associated with præcordial pain in 2 cases. In both of these old mitral disease was present, but there were no aortic lesions. Pain in the left side of the chest, or in the axilla, was complained of in 12 cases, and in all these the pleura on that side was inflamed as well as the pericardium. In 3 cases the pain was right-sided; all these were cases of right pleurisy.

The Temperature.—As would be expected, considering the various circumstances under which pericarditis may arise, no definite temperature chart can be deduced from the cases as a whole, but certain points with regard to the fever are evident. (1) The temperature was uniformly higher in the cases associated with pneumonia, 104° F. being a common maximum. (2) Of the cases judged to be rheumatic, the maximum temperature was on an average fully two degrees lower than in those due to pneumonia—i.e., about 102° F. In two cases, however, hyperpyrexia was present, and in four no fever. (3) In pyæmic cases the maximum temperature was high, ranging from 104° to 105° F. (4) The chronic renal cases form a class by themselves. In all save two the temperature was normal or subnormal. Even in the exceptional cases the temperature only rose on the day of death (102.6° and 103° F.), in each instance some days after the discovery of the pericardial friction. (5) The degree of pyrexia in no way served to indicate the nature of the pericardial contents. Fever was often as high when the effusion was serous as when it was purulent. Pus was occasionally present with a temperature which was subnormal or but slightly febrile.

The Pulse.—Owing to complications present in nearly all cases there is no direct evidence of the influence of pericarditis on the pulse-rate. The rate was rarely below 120, and terminal rates of 150 to 180 were registered. Twice the pulse was noted as intermittent, and in one of these the intermissions were respiratory in rhythm. It is noteworthy that in six of the renal cases the pulse-rate was not increased by the advent of pericardial inflammation.

Præcordial Bulging and Edema.—Edema of the præcordial region was not mentioned. In only one case was there evidence of bulging, and this was an infant in whom pericarditis was overlooked, but the left side of the chest at the nipple line measured one third of an inch more than the right. Edema of the legs and sometimes of the abdomen was noticed in 21 cases. In 13 this œdema was associated with valvular lesions, and in 7 with chronic renal disease. The remaining case was one of phthisis, with extensive lardaceous changes in the kidneys and other viscera.

Character of the Cardiac Impulse.—Of 59 cases where the character of the impulse was specifically noted, it was absent in 10, forcible in 5 (3 renal, 2 cardiac hypertrophy from mitral disease), and in the rest was noted "not strong," "feeble," "feeble and diffuse," "wavy." In 10 cases where the impulse could not be felt, the exudation was mainly plastic in 2; in 8 fluid effusion was present in amounts varying from a few ounces to fifty. In some cases it was bloody, in some serous, and in some purulent.

Pericardial Fremitus.—This is only mentioned in 10 cases, 4 of which were renal. In all the fatal cases but one the heart was hypertrophied and the amount of effusion not great. One was an operation case, where only 2 oz. of fluid were obtained.

Pericardial Fluctuation is not mentioned in any case.

Area and Shape of the Pericardial Dullness.—Owing to the frequency with which pericarditis was latent or terminal, there are only 39 cases out of the 112 in which accurate data with regard to the pericardial area are forthcoming. The most explicit accounts are in the cases of rheumatic origin. The pericardial areas were larger in this class than in the others, but the extent was no indication of the amount of fluid which is present, being largely due to the size of the heart. In many of these cases hypertrophy of the heart had long preceded the pericardial inflammation. In the group of cases where pericarditis complicated pneumonia accounts of increase in the area of cardiac dullness were lacking save in one instance, and in this one mediastinal infiltration was present, as well as pericarditis. In the terminal pericarditis of chronic renal disease (granular kidney) the dull area was, with one exception, evidently due to cardiac hypertrophy rather than to the amount of effusion.

The Upper Limit of Dullness.—Attention was directed to the records of upward extension of dullness along the left side of the sternum. In two cases the dull area passed above the second left costal cartilage. One of these was a case of granular kidneys with mitral obstruction, hypertrophied heart, and 18 oz. of serous effusion. The other was a case of rheumatic endocarditis in which fluid was encysted in the back of the sac, the remainder being obliterated

by adhesions. In 19 cases the dullness reached to the second left costal cartilage, but in 7 of those it was entirely cardiac. In the others varying amounts of effusion were present, mainly serous. The maximum amount in these cases was 55 oz. In 12 cases the dullness only reached the third left costal cartilage, and in these the quantity of fluid was insignificant.

The Right Limit of Dullness.—The maximum extension to the right was $\frac{1}{2}$ in. outside the right nipple. This occurred in the case of granular kidney, with 18 oz. of serum mentioned above. In 2 cases, both children and both rheumatic, the dullness reached to the right nipple; in one 20 oz. of fluid were found, in the other only 7 oz. In each case the heart was hypertrophied. In 13 cases the dullness was not traced beyond the right sternal edge, and in the rest it overstepped this by distances varying from $\frac{1}{2}$ in. to 2 in.

The Left Limit of Dullness.—Percussion dullness extended to the posterior axillary line in 1 case, which was one of the children mentioned in the preceding paragraph, a boy aged 11. Dullness reached into the axilla in 2 cases, and, curiously enough, in neither of these did it extend appreciably to the right of the sternum; in each instance the large area was due to enlarged heart rather than to excess of effusion. Dullness extended to the anterior axillary line in 3 cases; in all of these the heart was large, and the amount of effusion not great. The remaining cases, including those with the largest effusions, showed areas of dullness which extended from $\frac{1}{2}$ in. to $1\frac{1}{2}$ in. outside the left nipple.

Of Skiagraphic Examinations and Blood Counts there are no records.

Bacteriological Examinations.—*Staphylococcus albus* was recovered from the blood in 1 case of pericarditis with mitral disease. A streptococcus, probably pyogenes, was found in the blood from a case of malignant endocarditis and *Staphylococcus pyogenes aureus* was grown from an abscess in an obscure pyæmic case.

Condition of the Pleural Sacs.—A passive serous effusion existed in 16 cases. This effusion was left-sided in 2 cases (28 oz. and 16 oz.); right-sided in 3 cases (22 oz., 10 oz. and 2 oz.); bilateral in 11 cases, but in 6 of these the quantity was insignificant. In the remaining 5 the quantities were: Right 32 oz., left 12 oz.; right 10 oz., left 10 oz.; right 8 oz., left 10 oz.; right 35 oz., left 10 oz.; right 40 oz., left 40 oz. In none of these cases of passive serous pleural effusion did the pericardium contain pus, although its contents are described as turbid in 6 cases and blood-stained in 4. A purulent pleural effusion was present in 18 cases, and in 13 of these the pericardial fluid was also purulent, whilst in 2 it consisted of turbid serum, in 2 was blood-stained, and in 1 a myocardial abscess had led to rupture of the left ventricle. In 5 cases the empyema complicated lobar pneumonia and in 2 broncho-pneumonia. In 1 it resulted from pneumococcal abscess of the tibia, 4 were cases of pyæmia, 1 a case of malignant disease of the lung. In 5 cases the empyema appeared to be the primary disease. The empyema was right-sided in 5 cases, left-sided in 9 cases, bilateral in 4 cases. Of the primary empyemata

3 were left-sided, 1 right-sided, and 1 bilateral. Three of the primary empyemata were drained during life, three were overlooked.

Condition of the Lungs.—Clinically, pneumonia was recognized in 20 cases. In 13 of these the left lower lobe was the part involved, in 10 alone and in 3 in conjunction with other parts. The right lower lobe was consolidated in six cases, in 2 of these alone and in 4 in conjunction with other parts. There were also 3 cases in which pneumonia was present, but no notes made of the physical signs during life. Broncho-pneumonia was present in 8 cases, all infants or young children. Suppurating infarcts were present in 7 cases, all pyæmic. Massive collapse of the left lower lobe, uncomplicated by pleural effusion, occurred in 4 cases. The physical signs present were dullness near and below the lower angle of the left scapula, and tubular breath sounds in the same situation. In 1 case tubular breathing was also heard in the left axilla.

Treatment.—The general treatment consisted in the administration of salicylates to the rheumatic cases and the use of such cardiac stimulants as digitalis and strychnine. In a few instances opiates were administered. Leeches were applied to the præcordium in at least 11 cases. Blisters were only mentioned in 2 cases, but the records are probably at fault.

Operative Treatment.—In only 3 cases was operative treatment directed towards the pericardium; these cases were rheumatic. One was aspirated through the fourth left interspace, $4\frac{1}{2}$ oz. of blood-stained fluid being withdrawn. This patient had old mitral disease and died subsequently. The layers of the pericardium were then adherent. In a second case, portions of the sixth and seventh left costal cartilages were removed, the reflection of the pleura displaced, and 2 oz. of blood-stained serum evacuated. This patient made a good recovery. In a third case, the pericardium was aspirated through an incision in the subcostal angle. Between 4 oz. and 5 oz. of blood-stained serum were withdrawn, and a drachm of sterilized liquid paraffin injected into the sac with a view to prevent adhesions. This patient also recovered. Where empyema was recognized, it was invariably drained by rib resection. Five cases were treated in this way; in only one of them was the empyema right-sided. Serous pleural effusions were treated by aspiration, the right pleura being aspirated in 4 cases and the left in 3. Dry tapping is mentioned in 2 cases, each at the right lung base.

Mode of Death.—In most cases this appeared to be due to cardiac failure.

Conditions found at Post-mortem Examination.—The mediastinum was œdematous in 3 cases, inflamed in 1 case, the site of small sub-pleural abscesses in 1 case, indurated in 1 case, and infiltrated with growth in 2 cases. In cases where the pericardial exudate was purulent the sac was, with few exceptions, described as thickened and inflamed. In cases of serous effusion it was mostly described as inflamed and shaggy, but little mention being made of thickening.

Westminster Hospital.

By A. M. GOSSAGE, M.D., and H. F. MARRIS.

Statistics for sixteen years (1894 to 1909).

DURING the past sixteen years there have been 58 cases of pericarditis with effusion as determined by operation or post-mortem examination. The cases during successive years were as follows :—

1894 ... 2 cases	1900 ... 5 cases	1906 ... 3 cases
1895 ... 5 "	1901 ... 4 "	1907 ... 3 "
1896 ... 4 "	1902 ... 7 "	1908 ... 1 case
1897 ... 7 "	1903 ... 5 "	1909 ... 3 cases
1898 ... 3 "	1904 ... 2 "	
1899 ... —	1905 ... 4 "	

Of these patients, 37 were males and 21 females.

Aged under 1 year ... 6	Aged 30 to 40 years ... 4
„ 1 to 5 years ... 11	„ 40 „ 50 „ ... 9
„ 5 „ 10 „ ... 6	„ 50 „ 60 „ ... 4
„ 10 „ 20 „ ... 3	„ 60 „ 70 „ ... 1
„ 20 „ 30 „ ... 13	„ over 70 ... 1

Association with other Diseases.—In 20 patients the pericarditis was associated with pneumonia, of which 12 were cases of lobar pneumonia (4 left, 6 right, and 2 double); 8 patients had broncho-pneumonia. In 19 cases there was an empyema, in 10 of which no pneumonia was found after death. Thus more than half the cases were associated with pneumonia or empyema, and were probably pneumococic in origin. In 4 instances the pericarditis was associated with pulmonary tuberculosis, in 1 of which there was also pneumonia. In another of these the pericarditis was itself tubercular. In 1 case there was malignant disease of the lung which had spread to the pericardium and caused a purulent pericarditis. A gangrenous cavity at the base of the right lung had in another patient communicated with the pericardium. There were 3 cases of pyæmia, 1 of septicæmia, 1 of Ludwig's angina, 1 of erysipelas, 1 of diabetes, and 1 of Addison's disease. In 5 cases there were granular kidneys, 1 of which was associated with erysipelas (case mentioned above), and showed also purulent pericarditis and mediastinitis. In the other 4 no other accompanying morbid condition was found and the fluid in the pericardium was clear. Acute rheumatism was associated with the pericarditis in only 8 instances. The pericardial effusion was in 1 of these clear, in 4 sanious or blood-stained, in 2 turbid, and in 1 purulent. In several of these patients there was old-standing valvular disease.

Result, Course, and Duration.—Death resulted in all the cases here reported, and the course seems to have been progressively downhill. Many of the patients with pus in the pericardium were admitted moribund. From the first

discovery of pericardial rubbing until death in the rheumatic cases the duration varied from one day in a case with valvular disease and dropsy to twenty days. In the majority of the pneumonia and empyema cases the pericarditis was not diagnosed before death, and therefore it is impossible to estimate its duration. In those instances in which friction had been heard the duration varied from one day to thirty-eight days. A case of subphrenic abscess and purulent pericarditis lasted six days after pericardial friction had been first heard. In a case of erysipelas and granular kidney, pericarditis was diagnosed on the day of death. In another example of granular kidney, friction was heard fifteen days before death. A man, aged 73, was admitted with pleurisy and evidence of a large pericardial effusion (dullness up to the third rib and from the left nipple to the right); this patient lived fifteen days, and after death a pint of clear fluid was found in the pericardium. A woman with pyæmia after a miscarriage had signs of pericardial effusion on admission, which subsequently increased; the pericardium was opened and drained, and the patient survived the operation six days, dying twenty-one days after admission and two and a half months after the miscarriage.

Symptoms and Physical Signs.—In most of the cases there were no symptoms referable to the inflammation of the pericardium, or the symptoms were masked by grave intercurrent maladies such as pneumonia. In two of the rheumatic cases the aspect is said to have been anxious. Taking all cases, orthopnoea was noted 3 times; the posture was semi-recumbent in one case, on the back in 2, on the left side in 1, and in 1 instance the patient was stated to be unable to lie on the left side. Cyanosis was noted in 10 individuals, pallor in 6. In 13 cases marked dyspnoea was recorded, bad cough in 10. Vomiting was noted in 10 patients, severe in 3, moderate in 7. Pain was recorded as a prominent symptom in 10 instances: on the left side in 2 cases, right side in 2, in the chest in 1, in the abdomen in 1, and præcordial pain in 3, in 2 of which there was also præcordial tenderness. It must be remembered that many of these patients were suffering with pneumonia or pleurisy. The pulse-rate was usually increased and varied between 180 and 80 in different individuals. As a rule the pulse was regular, but in 3 of the more infrequent examples it was irregular, and in 1 case of pneumonia the pulse was 144 and intermittent. The temperature was raised in all cases except 2. In 3 instances it was subnormal before death; the highest temperature recorded was 108° F. just before death. In those cases of pericarditis in which there was no pneumonia the temperature varied between 100° F. and 101° F. The character and position of the cardiac impulse naturally varied considerably with the degree of dilatation of the heart. It was recorded as being outside the left nipple in 5 cases, in the nipple line in 2, and inside the nipple line in 2. In the vast majority of patients the heart was not examined on the day of their death. Neither fremitus, præcordial bulging, œdema, nor fluctuation seem to have been noticed in any of the cases. The cardiac dullness was not increased in 2 cases, increased in 11, in several of which there was valvular disease and dilatation of the heart. The superficial dullness was up to the third rib in several

patients, up to the second rib in only 1. The widest area of superficial dullness recorded extended from the left nipple to the right nipple, and in this case a pint of fluid was found in the pericardium after death. In 7 cases the dullness extended to the right sternal edge or beyond, and in two instances to the mid line of the sternum. Pericardial friction was noted in 8 cases, whilst in 3 others it was specially stated to have been absent. The heart sounds were distant or feeble in 6 cases, inaudible in 1 case. The sounds were normal in 5 cases, and in 1 case were so loud that they could be heard all over the chest. The sounds were once described as tic-tac. A systolic apical bruit was heard in 7 cases and double aortic bruits in 3 cases.

Condition of Pleura.—As mentioned above, empyema was present in 19 patients, 9 on the left side, 5 on the right and 5 double; 6 of these cases were operated on. Clear fluid was present in the pleura in 12 cases, in 2 of which the fluid was blood-stained.

The blood was only examined in one case. In this, the first leucocyte count showed 11,000, of which 75 per cent. were polymorphonuclear, and at this time an exploration of the pleura gave only clear fluid. Eighteen days later the leucocyte count was 39,000, of which 85 per cent. were polymorphonuclear. The right pleura was then opened and a pneumococcic empyema drained.

In only one case was the pericardium opened and drained. This was that of a woman, aged 34, who was attacked with pyæmia after a miscarriage. On admission in 1901 there were signs of pericardial effusion. The patient was treated with antistreptococcic serum and at first improved. Later the pericardial effusion increased and abscesses developed in the right shoulder and right hip. The cardiac dullness extended from the anterior axillary line on the left to 1 in. beyond the right edge of the sternum, and up to the second rib. The heart sounds were very indistinct. The abscesses were opened and the pericardium was opened and drained by removing portions of the fourth and fifth costal cartilages. The walls of the pericardium were very thick and a considerable amount of pus was evacuated, which gave a pure growth of *Staphylococcus pyogenes aureus*. After the operation the patient improved for a short time, but later became weaker and died six days after the operation. A man aged 30 was admitted with angina Ludovici. The neck was incised, and in the pus so obtained streptococci were found. Antistreptococcic serum was injected, but the patient died forty-eight hours after admission. Post-mortem purulent mediastinitis and pericarditis were found.

Post-mortem Findings.—The mediastinum was found inflamed in 10 instances, in 4 of which pus was found in the tissues. In all these cases, except one, purulent pericarditis was also present; in the exceptional case the nature of the fluid in the pericardium was not mentioned. The mediastinal glands were enlarged in 10, in one of which the glands were tuberculous, in another infiltrated with malignant growth. In all the 58 cases there was evidence of pericardial inflammation. In 23 the fluid effusion was purulent.

and in 23 turbid. Of the latter, many were probably becoming purulent; but in some of them it is specially stated that, though turbid, the fluid was not purulent. The quantity of fluid varied from a small amount up to what produced great distension of the sac. Where a definite amount was mentioned the largest quantity was 1 pt. of clear serous fluid. The largest amount of pus mentioned was 5 oz., but in 7 other cases the pericardium was said to be distended with purulent fluid. In the majority of the cases the surface of pericardium was covered with shaggy lymph. The effusion was in several cases blood-stained, both when the fluid was purulent and when it was clear. In one patient the pericarditis was tuberculous, and in this the effused fluid was clear. In another the pericarditis was due to a malignant growth spreading to the pericardium from the lung: here the effusion was turbid. Valvular disease of the heart was found in 13 bodies, of which 7 showed recent vegetations. There was myocarditis in 1 case, and the myocardium was described as soft in 2 cases. In most of the patients the liver, spleen, and kidneys were febrile, in 9 cases the liver was nutmeg or cardiac. In one person there were miliary tubercles in the liver, and in another in the kidneys. There were both perisplenitis and perihepatitis once, once perisplenitis alone, and once perihepatitis alone. The kidneys were granular in 5 cases. In two examples of pyæmia there were abscesses in the kidneys. A case of broncho-pneumonia, associated with empyema, had also purulent pericarditis, peritonitis, and meningitis.

Bacteriology.—Bacteriological examination of the pus from the pericardium showed the presence of the pneumococcus in 6 cases and of a diplococcus, which was probably the pneumococcus, in one. Staphylococci were found in 3 cases and streptococci in 1. In the associated empyemata pneumococci were found once and staphylococci once. In a case of pyæmia with purulent pericarditis, streptococci were found in the pus obtained from the knee-joint and from vegetations on the aortic valves. In a patient with Ludwig's angina streptococci were obtained from the tissues of the neck; antistreptococcic serum was injected without relief. A youth aged 18 developed pyæmia and purulent pericarditis after acute osteomyelitis of the tibia: his blood gave a growth of staphylococci. The leg was amputated and anti-staphylococcic serum injected, but the patient died eight days after admission.

Royal Free Hospital.

By IVY E. WOODWARD, M.D.

Statistics for fifteen years, from June 30, 1894, to June 30, 1909.

No cases have been treated by operation during this period. Number of cases, 50.

SUMMARY OF THE FIFTY CASES.

Case	Age	Sex	Concurrent illness	Quantity of effusion (in ounces)	Nature of effusion
1	18	M.	Chronic valvular disease, acute endocarditis	3	Not noted
2	19	F.	Chronic valvular disease	19	Clear yellow
3	17	M.	Acute rheumatism, acute endocarditis	2	Not noted
4	About 60	M.	Granular kidneys, pleurisy	1½ to 2	Turbid, semi-purulent
5	20 mos.	M.	Pneumonia	2 to 3	Purulent
6	21	F.	Acute rheumatism, chorea, acute endocarditis	8	Clear
7	29	F.	Lobar pneumonia	Over 10	Not noted
8	19	M.	Acute rheumatism, acute endocarditis	10	Not noted
9	25	M.	Lobar pneumonia	2½	Serous
10	58	M.	Chronic valvular disease, acute endocarditis	3	Clear yellowish
11	36	M.	Chronic interstitial nephritis	About 1	Semi-purulent
12	15	F.	Small white kidneys, uræmia	4	Sero-fibrinous, with a large quantity of stringy lymph
13	6 weeks	M.	Pneumonia	"A small excess"	of fluid with large flakes of lymph
14	25	F.	Lobar pneumonia	Not noted	Clear yellow
15	33	M.	Lobar pneumonia	Full of clear fluid	
16	30	M.	Mitral stenosis	7	Slightly turbid with a few fragments of lymph
17	31	M.	Lobar pneumonia, purulent pleurisy	5 to 6	Purulent
18	11	F.	Chorea, acute rheumatism	9	Blood-stained, slightly turbid
19	47	M.	Lobar pneumonia	3 to 4	Thin, milky, semi-purulent
20	39	M.	Lobar pneumonia	2	Slightly turbid
21	4 mos.	F.	Broncho-pneumonia	1	Turbid
22	5	M.	Pulmonary tuberculosis, meningitis	5	Semi-purulent, with flakes of lymph
23	36	F.	Chronic endocarditis, acute endocarditis	6	Clear, straw-coloured
24	10 mos.	F.	Pneumonia	3	Yellowish, sero-purulent
25	59	M.	Double empyema, suppurative mediastinitis	2	Serous
26	35	F.	Alcoholic polyneuritis, general miliary tuberculosis	1½	Clear
27	31	M.	Hydronephrosis	4	Blood-stained

SUMMARY OF THE FIFTY CASES (*continued*).

Case	Age	Sex	Concurrent illness	Quantity of effusion (in ounces)	Nature of effusion
28	9	F.	Acute infective osteomyelitis	3	Sero-purulent, containing staphylococci
29	3	M.	Acute infective periostitis	6	Purulent, staphylococci
30	5	M.	Pyæmia	3	Blood-stained, sero-purulent, growing <i>Staphylococcus pyogenes aureus</i>
31	2	M.	Broncho-pneumonia	Not noted	Purulent fluid
32	2	M.	Septicæmia	1 or more	Clear
33	10 mos.	F.	Lobar pneumonia	Several	Turbid, yellow, containing pneumococci
34	25	M.	Pneumonia, empyema, pneumothorax	Full of fluid,	containing flocculi of lymph
35	16	M.	Chronic valvular disease, acute endocarditis	10	Clear, blood-stained
36	4 weeks	M.	Pyæmia	1 drachm	Purulent
37	15	F.	Chronic valvular disease	A little	Clear, containing flakes of lymph
38	31	F.	Chronic valvular disease	2	Slightly turbid
39	41	F.	Chronic valvular disease	2	Turbid, yellow, containing flocculi of lymph
40	20	M.	Malignant endocarditis	4	Clear, straw-coloured
41	3	M.	Lobar pneumonia, empyema	$\frac{1}{2}$	Thick, yellow, purulent
42	11	M.	Acute infective osteomyelitis	$\frac{1}{2}$	Turbid
43	12	F.	Acute infective periostitis	30	Blood-stained, purulent
44	29	M.	Acute endocarditis	Some excess	Clear
45	20	M.	Chronic valvular disease, acute endocarditis	8	Clear yellow
46	16	F.	Acute endocarditis	2	Blood-stained
47	9	M.	Chronic valvular disease	12	Clear yellow, some flakes of lymph
48	24	M.	Pyonephrosis	28	Clear yellow, slightly blood-stained
49	5	M.	Empyema, malignant endocarditis	About 2	Slightly turbid, straw-coloured
50	20 mos.	F.	Broncho-pneumonia	—	Pericardium distended and full of pus

London Hospital.

By H. L. TIDY, M.B., R. S. WOODS, M.D., and C. E. ZUNDEL, M.D.

IN the London Hospital, between the years 1893 and 1908, pericarditis with effusion occurred in 121 cases in which the diagnosis was confirmed by post-mortem examination. This number excludes all those in which the amount of fluid present did not exceed 2 oz. The conclusions drawn from the analysis of this series will be presented under the following headings: (1) *Ætiology*, (2) *Disease Association*, (3) *Symptoms*, (4) *Signs*, (5) *Course*, (6) *Morbid Anatomy*.

(1) ÆTIOLOGY.

Age.—The age and sex-incidence is shown in the following table :—

TABLE I.

	Male	Female	Total
Aged under 1 year	4	5	9
„ 1 to 5 years	14	2	16
„ 5 „ 10	8	7	15
„ 10 „ 20	13	14	27
„ 20 „ 30	15	5	20
„ 30 „ 40	11	4	15
„ 40 „ 50	9	2	11
„ 50 „ 60	5	1	6
„ 60 „ 70	1	1	2
	80	41	121

The disease incidence, therefore, rises from infancy until it attains a maximum between the ages of 10 and 20, from this period showing a gradual fall in successive decades. The relation to age of pericarditis with effusion is determined by the frequency of certain diseases which will be referred to below. The primary cause varies at different ages, and the maximum which is found to occur between the ages of 10 and 20 is due to the frequency of both rheumatism and pneumonia at this period.

(2) DISEASE ASSOCIATION.

(a) *Primary Pericarditis.*—In 3 cases it was noted that no other organic disease was present. In one of these the effusion was purulent.

(b) *Secondary Pericarditis.*—The following are the associated diseases arranged in order of frequency: Rheumatism and chorea, pneumonia, renal, empyema, heart disease, broncho-pneumonia, septicæmia and pyæmia, other diseases. The occurrence of these diseases at the various ages is shown in the following table :—

TABLE II.

Years	Broncho-pneumonia	Empyema	Pneumonia and pleurisy	Rheumatism and chorea	Morbus cordis without rheumatism	Renal	Septicæmia and pyæmia
— to 1	6	3	—	—	—	—	—
1 „ 5	5	6	2	2	—	1	—
5 „ 10	—	—	—	9	2	—	1
10 „ 20	—	2	4	14	4	—	2
20 „ 30	—	2	7	1	5	2	2
30 „ 40	—	—	3	—	1	7	4
40 „ 50	—	—	5	—	—	6	—
50 „ 60	—	—	2	2	—	—	—
60 „ 70	—	—	1	—	—	1	—

All the cases, therefore, under one year occurred in association with broncho-pneumonia or empyema. Subsequently the most important factor is found to be, successively, rheumatism, pneumonia, and chronic kidney disease. The actual percentages are given in Table III.

TABLE III.

Age	Broncho-pneumonia and empyema (per cent.)	Rheumatism and heart disease (per cent.)	Pneumonia and empyema (per cent.)	Renal (per cent.)
Under 1 year ...	100	—	—	—
1 to 5 years ...	68·7	12·5	12·5	6·3
5 „ 10 „ ...	—	73	—	—
10 „ 20 „ ...	—	68	21	—
20 „ 30 „ ...	10	30	45	10
30 „ 40 „ ...	—	6·7	20	47
40 „ 50 „ ...	—	—	44	54

From these two tables it is clear that: Under 1 year, broncho-pneumonia or empyema is present in every case. Between 1 and 5 years, broncho-pneumonia or empyema is present in nearly three-fourths of the cases. Between 5 and 10 years, rheumatism and heart disease now account for a similar proportion, whilst pulmonary causes are absent. Between 10 and 20 years, rheumatism and heart disease account for a still larger number, but the percentage shows a fall owing to the commencing importance of pneumonia as a factor. Between 20 and 30 years, pneumonia attains its greatest influence, whilst that of rheumatism becomes less marked. Between 30 and 40 years, kidney disease occurs in half the cases. Between 40 and 50 years, kidney disease accounts for an even higher percentage. Above 30 years the greater number of cases occurred in males, as one would expect from the influence of renal causes.

There was no case recorded in association with intrathoracic neoplasm.

(3) SYMPTOMS.

It is difficult to estimate the influence of the intercurrent disease in the causation of symptoms. Marked dyspnoea was recorded in more than 50 per cent; vomiting occurred in 30 per cent.; severe præcordial pain in only 13 per cent. Pain, therefore, was not of frequent occurrence. Restlessness and delirium were rarely present.

(4) PHYSICAL SIGNS.

Pallor was extreme in 26 cases. Cyanosis, apart from pulmonary complications, was absent. The temperature was subnormal in 6 cases, under 100° F. in 26 cases, 100° F. to 102° F. in 42 cases, 103° F. to 104° F. in 38 cases, and above 104° F. in 2 cases. The highest recorded temperature was 105° F. No case of extreme hyperpyrexia occurred.

Pulse.—The rate was invariably increased, in only twelve cases being under a hundred.

Area of Cardiac Dullness.—Lateral enlargement alone seems to have been more common than the triangular area of dullness usually described. In 6 cases only did the upper limit reach the second rib.

Pericardial Friction.—This was detected in 32 cases at some period of the disease.

Heart-sounds.—In the majority of cases no "muffling" of the heart-sounds was noted.

(5) COURSE.

The duration in those cases in which it could be estimated with sufficient accuracy was as follows :—

Under 1 week	33 cases
" 3 weeks	39 "
Above 3 "	23 "

The shortest duration was three days.

(6) MORBID ANATOMY.

(1) *Amount of Fluid.*—The amount of fluid found post mortem was :—

Under 3 oz.	23 cases
3 to 10 oz.	54 "
Over 10 oz.	44 "

The largest amount of fluid was 2 pints, this quantity occurring in 2 cases.

(2) *Nature of Fluid.*—The cases have been grouped according as the fluid was: (a) Serous, (b) blood-stained, (c) purulent.

(a) *Serous.*—Under this heading are included those cases in which the fluid was described as "turbid."

(b) *Blood-stained.*—This group is a small one composed of only 9 cases, 3 of which occurred in association with chronic nephritis.

(c) *Purulent.*—The fluid was purulent in 35 cases, in one of which the presence of blood was noted.

Under 1 year	6 cases
1 to 5 years	10 "
5 " 10 "	1 case
10 " 20 "	5 cases
Above 20 "	11 "

The disease association is shown in the following table :—

Pneumonia and broncho-pneumonia	19 cases
Empyema (right)	4 "
" (left)	6 "
Septic conditions	5 "

Under the heading of pneumonia and broncho-pneumonia are included 3 cases of broncho-pneumonia with measles and one of tuberculosis. The septic conditions include pyæmia, septicæmia, and 1 case of hydatid abscess. In 1 case the condition was apparently primary. There was no instance of definitely purulent pericarditis in association with rheumatism. The average amount of pus was 10 oz. and the maximum 2 pints.

Victoria Hospital for Children, Chelsea.

By H. F. L. HUGO, M.B.

NUMBER of cases 15, out of 650 post mortems. Number in each year:—

1894 (half year)	—	1900	...	1	1906	...	—
1895	...	1901	...	—	1907	...	2
1896	...	1902	...	—	1908	...	3
1897	...	1903	...	1	1909 (half year)	...	1
1898	...	1904	...	1			
1899	...	1905	...	2			

Age	Sex	Previous illness	Concurrent illness	Duration
10	F.	Measles as a baby	Double mitral	—
2	M.	Bronchitis five months previously	Nil	—
11	F.	Scarlet fever when aged 3, followed by morbilli and pertussis; patient in St. George's Hospital (cough)	Double mitral	—
10	M.	Measles as a baby	Spinal caries and pyæmia, abscess in ankle and spine	—
9	M.	Pertussis and measles	Nil	6½ months
6	F.	Pertussis and measles three years ago	Psoas abscess, broncho-pneumonia	—
21 months	M.	Nil	Nasal diphtheria	—
7	F.	Measles two years ago, varicella one year ago	Nil	28 days
7	F.	Nil	Acute rheumatism, mitral regurgitation, varicella	—
15 months	M.	Nil	Pneumonia	6 weeks
14 months	M.	Measles three weeks previously	Empyema	—
11	M.	Not obtained	Mitral regurgitation	—
4	M.	Measles when aged 2	Acute necrosis of left clavicle	3 days
9	F.	Nil	Bronchitis	3 months
15 months	F.	Measles when aged 3 weeks	Fibroid lung	—

No.	Result	Aspect	Posture	Cyanosis	Pallor	Dyspnoea
1	Death	Pale	Lies down comfortably	Yes	Yes	Yes
2	"	—	—	—	—	—
3	"	Pale	Orthopnoea	—	Yes	"
4	"	—	—	—	—	—
5	"	Veins of neck distended	Lies down	Yes	—	Yes
6	"	Flushed face	—	No	No	"
7	"	Pale	—	—	Yes	"
8	"	Anxious expression	—	—	—	Slight
9	"	—	—	—	—	Yes
10	"	—	—	No	Yes	"
11	"	Collapsed	—	—	"	"
12	"	—	—	Slight	No	"
13	"	—	—	Yes	—	"
14	"	—	—	"	No	"
15	"	Listless	—	—	Yes	"

No.	Cough	Vomiting	Pain	Tender- ness	Œdema	Pulse	Temperature	Præcordial bulging
1	Slight	Persistent last 2 days	Yes, over heart	No	None	Small and irregular	97° to 101°	Slight
2	No	Yes	—	—	—	112 to 156	97° to 101°	No
3	—	—	Yes, over heart	—	Yes, legs and ascites	Rapid and irregular	100°; 96° before death	Marked
4	—	—	Back of neck	—	None	98 to 144	95° to 102°	—
5	Yes	No	No	No	Yes, all over body	Feeble	—	No
6	—	—	—	—	—	Weak, 160	102° to 106°	—
7	—	—	—	—	—	Rapid	97° to 101°	—
8	—	No	—	—	—	Weak, 160	97° to 102°	No
9	—	—	Yes, over præcordium	—	No	100 to 150	97° to 103°	—
10	—	Yes	—	—	Yes, feet and ascites	160	97° to 103°	—
11	No	—	—	—	No	120 to 140	97° to 103°	—
12	Yes	No	—	—	—	130, low tension	98° to 103°	—
13	No	Once	—	—	—	160	104°	No
14	Yes	Yes	No	No	Yes, feet	Weak, beats dropped	98° to 103°	Slight
15	—	—	—	—	No	130 to 160	97° to 102°	—

No.	Præcordial œdema	Impulse	Position of impulse	Fremitus	Fluctua- tion	Area of dullness
1	No	Weak and diffuse	Fourth space in N.L.	No	No	First left space, right ster- nal line, 1 in. outside N.L.
2	No	—	—	—	—	—
3	—	Forcible	—	Faint	—	Right sternal line, 3 finger- breadths outside N.L.
4	—	—	—	—	—	—
5	No	Not felt	—	No	No	Second left space; no dullness to right of ster- num, 1½ in. outside N.L.
6	—	—	Fifth space in N.L.	—	—	—
7	—	—	—	—	—	Third space, mid-line, ½ in. outside N.L.
8	No	Diffuse	—	Yes	—	First rib, right N.L., 1 in. outside N.L.
9	No	Fluttering	Fifth space	No	No	Left sternal line, A.B.
10	—	—	Fourth space, 2 in. outside N.L.	—	—	—
11	—	—	—	—	—	—
12	—	Faint	Fifth space in N.L.	—	No	Third left space, right edge of sternum, N.L.
13	—	—	—	—	—	—
14	—	—	Fifth space, 1 in. outside N.L.	No	No	Clavicle, right edge of sternum, 1 in. outside N.L.
15	—	—	—	—	—	—

Influence of change of posture, no information.

No.	Auscultation of pericardium	Auscultation of heart	Blood	Pleura
1	Friction rub over lower part of sternum, later as far out as A.B.	Apex, systolic and presystolic; pulmonary systolic and loud second sound	—	<i>Nil</i>
2	<i>Nil</i>	<i>Nil</i>	—	—
3	Friction rub over base and down sternum	Double murmur at apex	—	<i>Nil</i>
4	<i>Nil</i>	<i>Nil</i>	—	—
5	No friction rub	Heard as faint taps in third and fourth spaces	—	Effusion both sides
6	<i>Nil</i>	<i>Nil</i>	—	<i>Nil</i>
7	"	"	—	"
8	To-and-fro rub over cardiac dullness	Apex, systolic conducted out; pulmonary, second sound loud	—	"
9	Friction rub at base	Apex, systolic conducted out; pulmonary, second sound loud	—	"
10	<i>Nil</i>	<i>Nil</i>	Red, 3,500,000 White, 24,000	"
11	"	"	—	Empyema, left base
12	To-and-fro friction rub	Apex, systolic conducted out	—	<i>Nil</i>
13	<i>Nil</i>	<i>Nil</i>	—	"
14	"	Systolic and presystolic	No organisms found	"
15	"	<i>Nil</i>	—	"

No.	Condition of lung	TREATMENT		Operation
		General	Local	
1	Note high-pitched in left axilla	Stimulant	Ice-bag, later, blisters	None
2	Dullness at left base behind, tubular breathing, bronchophony, fine crepitations	"	<i>Nil</i>	"
3	<i>Nil</i>	"	Leeches	"
4	A few râles at bases	—	<i>Nil</i>	"
5	Pleural effusion	—	"	"
6	Consolidation of left lower lobe	Stimulant	Poultice to left side	"
7	Left, nil; right, broncho-pneumonia	"	<i>Nil</i>	"
8	Lungs clear	"	Leeches, blister	"
9	Dullness and tubular breathing at left side	"	Leeches	"
10	Consolidation of right upper lobe	"	—	"
11	<i>Nil</i>	—	<i>Nil</i>	"
12	Dullness and diminished breath sounds at right base	—	Blister	"
13	Impaired resonance and crepitations at both bases	—	<i>Nil</i>	"
14	Bronchitis	Antistreptococcic serum	—	"
15	Dullness, tubular breathing, and crepitations over left lung	Stimulant	Poultice	"

No record of any skiagrams.

POST MORTEM.

No.	Mediastinum	Pericardium	Quantity of fluid	Nature of fluid
1	Glands enlarged	Distended, thickened, adherent to heart and lungs	3½ oz.	Clear, straw-coloured
2	Glands enlarged	Distended, adherent at base, shaggy with lymph	2 to 3 oz.	Turbid
3	and caseous Nil	Old adhesions over left ventricle, recent adhesions over right ventricle	Quantity	Blood-stained
4	„	Distended with air and fluid, lymph on surface, visceral surface hæmorrhagic	2 oz.	Sero-purulent
5	Glands enlarged	Thickened, adherent at base and apex, lymph on surfaces	6 oz.	Thin, purulent, with masses of coagulum
6	„	Recent lymph over both surfaces, adhesions at base	3 oz.	Slightly turbid
7	Glands caseous	Distended lymph on surfaces	Large quantity	Fibrinous, muddy, streptococcus
8	Nil	Much enlarged, adherent to epicardium, with pockets of pus in between	—	Blood-stained, serous fluid
9	Glands enlarged and œdematous	Greatly distended, both layers adherent at apex and to chest wall	Large quantity	Clear, serous fluid, with flakes of lymph
10	Nil	Thickened, adherent to epicardium by thick adhesions	½ oz.	Semi-purulent
11	„	Distended, adherent to chest wall	Large quantity	Purulent, streptococcus
12	Glands enlarged	Shaggy lymph over both surfaces, recent adhesions	4½ oz.	Deeply blood-stained
13	Nil	Shaggy lymph	Several oz.	Purulent
14	Glands enlarged	Encasing blood-stained lymph	2½ oz.	Blood-stained, almost clear
15	„	Thickened and covered with lymph	1 oz.	Semi-purulent

No.	Heart	Fluid in pleura	Fluid in peritoneum	Blood
1	Left ventricle and auricle slightly hypertrophied, vegetations on mitral valve	3 oz. clear fluid in each pleura	Small quantity	—
2	Nil	None	—	—
3	Muscle pale and friable, mitral stenosis, aortic cusps thickened	¾ pint clear fluid	None	—
4	Muscle pale, right side dilated	Small quantity	—	—
5	Mitral valves thickened, old vegetations	Nil	Nil	—
6	Muscle good, valves healthy	None	„	—
7	Normal	Small quantity clear fluid	„	—
8	Both ventricles hypertrophied, mitral valves dilated, and vegetations at base	5 oz. serous fluid and lymph	5 oz. clear	—
9	Mitral endocarditis, fatty degeneration of heart muscle, ventricular hypertrophy (left)	Clear fluid in each pleura	Small quantity	—
10	Left ventricle hypertrophied, valves nil	Nil	1 pint, semi-purulent	—

POST MORTEM (continued).

No.	Heart	Fluid in pleura	Fluid in peritoneum	Blood
11	Healthy	—	—	—
12	All cavities dilated, mitral valves thickened, aortic, granulations on edge of cusps	4 oz. clear (right) 3 oz. clear lymph (left)	4 oz. clear	—
13	Normal	Nil	Nil	—
14	Right side dilated, left dilated and hypertrophied, mitral contracted and recent vegetations, aortic recent vegetations	„	1 pint clear	—
15	Left ventricle hypertrophied, valves healthy	—	A little pus in pelvis	—

No.	Lungs	Liver	Spleen	Kidneys
1	Congested	Enlarged, dark	Enlarged, otherwise normal	Slightly congested
2	Abscess in left base, both congested and showed tubercle bacilli	Normal	Tubercles	Normal
3	Adhesions at left apex and left base, bronchitis at right lower lobe	Nutmeg	Cardiac	Cloudy swelling and cardiac
4	Collapsed at bases, hypostatic congestion	Fatty	Enlarged, dark, numerous recent infarcts	Cloudy swelling
5	Left upper lobe fibroid, rest of both lungs emphysematous, and patches of broncho-pneumonia	Nutmeg, enlarged	Cardiac perisplenitis	Normal
6	Left, recent pleurisy, lower lobe consolidated, upper emphysematous; right, lower engorged, recent pleurisy, upper congested	Nil	Nil	Nil
7	Adhesive pleurisy, numerous small caseous nodules in lungs	Miliary tubercle bacilli	Small, caseous nodules	Congested, no tubercle bacilli
8	Both congested	Slightly enlarged	Slightly enlarged	Congested
9	Both œdematous, adhesive pleurisy right side	Enlarged, and slight degree of cyanosis	Slightly enlarged and soft	Congested
10	Adherent pleurisy both sides; right, old pneumonia upper lobe	Nil	Nil	Nil
11	Left collapsed, left adherent	Nil	Enlarged	Nil
12	Congested	Enlarged, nutmeg	Soft	Pale
13	Acute congested	Slightly congested	Slightly congested	Slightly congested
14	Areas of consolidation and collapse over both lungs, especially right	Slightly enlarged, nutmeg in places, pale	Normal	Rather pale, otherwise nil
15	Right, miliary tubercle bacilli and caseous broncho-pneumonia; left, adherent, upper lobe caseous with cavity, lower miliary tubercle bacilli	Fatty, numerous miliary tubercle bacilli	Nil	Congested

No cytological or bacteriological examinations of fluids or blood.

East London Hospital, Shadwell.

By J. SIDNEY PEARSON, M.D.

Statistics for ten years (1899 to 1909).

NUMBER OF CASES, 53.

Associated with empyema	35	} All purulent.
„ „ pneumonia without empyema	6	
„ „ pyæmia	4	
„ „ tuberculosis	2	
„ „ traumatism	1	
Associated with rheumatism and morbus cordis	5	Non-purulent.
	<hr/> 53	

CASES DIAGNOSED OR SUSPECTED.

	Diagnosed	Suspected	Unsuspected
35 with empyema	7	6	22
6 pneumococcal	2	—	4
4 pyæmic	2	—	2
1 traumatic	1	—	—
2 tuberculous	—	—	2
5 rheumatic	5	—	—
<hr/> 53	<hr/> 17	<hr/> 6	<hr/> 30

SIX CASES OF PYO-PERICARDITIS ASSOCIATED WITH PNEUMONIA BUT WITHOUT EMPYEMA.

	Sex	Age in Years	Amount of pus	Bacteriology	Pneumonic lesion	
1	M.	6	2 oz.	Pneumococci	Double basal	Unsuspected; undiagnosed
2	M.	1½	1 oz.	Pneumococci, streptococci	Left base	Undiagnosed
3	M.	1¼	4 to 6 oz.	Pneumococci	Right base	Empyema suspected; leucocytes 48,000, later 56,000; explored three times without result; X-rays, <i>nil</i> definite
4	M.	1½	5 to 6 oz.	Pneumococci and motile bacillus like <i>Bacillus coli communis</i>	Not discovered; ? primary pericarditis	Suspected, but no increase of cardiac dullness made out; Heart sounds distant; H.A.B. not felt
5	F.	1	5 oz. turbid fluid	Pneumococci	Meninges; peritoneum	Suspected; cardiac dullness increased to left anterior axilla line; H.A.B. invisible
6	F.	4½	1 oz.	Pneumococci	Peritoneum and left base	Admitted moribund; no time for diagnosis

TWO CASES ASSOCIATED WITH TUBERCULOSIS.

	Sex	Age in years	Amount of pus	Bacteriology	Pneumonic lesion	
1	F.	3	2 oz.	Sterile	Caseous cervical glands (cause of admission); caseous bronchial glands; caseous mesenteric glands; caseous lungs	Death very sudden; undiagnosed
2	M.	9	1 oz.	Sterile	General caseous tuberculous lungs, mesentery, &c.	Admitted for general oedema; undiagnosed

ONE CASE ASSOCIATED WITH TRAUMATISM.

Girl, aged 8; run over. Fractured eighth rib, left side; rapid necrosis with abscess (staphylococci). Three days later marked pericardial friction; died twelve hours later, suddenly. 3ij staphylococcal pus in pericardium. Direct extension from necrosed rib traced. Diagnosed.

FOUR CASES ASSOCIATED WITH PYÆMIA.

	Sex	Age in years	Amount of pus	Bacteriology	Primary lesion	Pneumonic lesions
1	M.	7	About 1 oz.	<i>Staphylococcus aureus</i>	Osteomyelitis; right, humerus, left, femur	Multiple abscesses: kidneys, liver, spleen, myocardium; undiagnosed
2	M.	1½	About 2 oz.	Not stated	Abscess of face; ? alveolar	Diagnosed before death
3	M.	2	4 to 5 oz.	<i>Staphylococcus aureus</i>	Osteomyelitis; right femur	Multiple abscesses in kidneys; diagnosed before death, marked friction and general increased cardiac dullness
4	F.	6	1 oz.	Pneumococci	Multiple boils	Multiple abscesses: kidneys, lungs, and mastoid; 1 oz. of pus in left pleura; undiagnosed

FIVE CASES ASSOCIATED WITH RHEUMATISM.

No.	Sex	Age in years	Quantity of fluid	Points in diagnosis	Other remarks
1	F.	12	6 to 7 oz. clear straw; sterile	Early pericardial rub, later sounds muffled; extreme cyanosis; cardiac dullness one inch to right of sternum	Died shortly after paracentesis pericardii; 15 oz. of straw fluid in each pleural sac; old and recent endocarditis and vegetations
2	F.	12	"Enormously distended;" straw fluid	H.A.B. fifth space mid-axillary line; cyanosis and dyspnoea: no enlargement to right made out; marked friction sounds	Heart weighed 18 oz.; old endocarditis mitral valve; left vent. hypertrophied
3	F.	8½	10 oz. blood-stained, adherent at apex	Increased dullness and friction; diffuse H.A.B.; dyspnoea	Pericardium extended to right nipple and up to first rib; recent vegetations on all valves; first attack of rheumatism; double pleural effusion
4	F.	11	Collection, straw-coloured on right side; rest adherent; sterile	Increased cardiac dullness; dyspnoea; palpitation; friction of heart followed by muffled sounds	Ascites; second attack
5	F.	7	6 oz. clear yellow	Dyspnoea; cardiac dullness to right of sternum; irregular heart and faintly heard	Operation for draining pericardium; died before sac was opened; 1 pt. of clear fluid in each pleural sac

THIRTY-FIVE CASES ASSOCIATED WITH EMPYEMA.

Sex :—Males, 22; Females, 13.

Age :— Under 1 year (youngest 5 months) 6
Over 1 year and under 2 years 15
Over 2 years and under 5 years 12
Over 5 years and under 10 years (oldest 7 years) 2

Bacteriology (26 cases examined) :—

Pneumococci only 20
Pneumococci, with streptococci and *Bacillus coli communis* 1
Pneumococci with streptococci 1
Streptococci only 1
Bacillus influenzae only 1
Reported sterile 2

Empyema :— Both sides 9 cases
Right side 12 „
Left side 14 „

THIRTY-FIVE CASES ASSOCIATED WITH EMPYEMA.

No.	Sex	Age in years	Amount of pus in pericardium	Bacteriology	If diagnosed	Side of empyema	Cardiac dullness	Friction	
1	F.	7	4 oz.	Pneumococci	Yes	Right, 1½ pt. pus	+	+	—
2	M.	3½	1 oz.	Pneumococci	No	Right and left, 1½ oz. pus	No	—	Pneumococcal peritonitis
3	F.	4½	2 oz.	Pneumococci	No	Left, 6 oz.	No	—	—
4	M.	1	¾ oz.	Pneumococci	No	Left, 12 oz.	No	—	—
5	M.	9 months	1 oz.	Pneumococci	Suspected	Right and left	+	—	Inspissated pus in pleura
6	M.	18 months	1 oz.	—	Yes	Left	+	Distant	—
7	M.	14 months	1½ oz.	Pneumococci	No	Left, 2 oz.	No	—	—
8	F.	2	1½ oz.	—	No	Right and left	—	—	—
9	M.	14 months	1 oz.	—	Suspected	Left, 4 oz.	+	+	—
10	M.	1	2 oz.	—	No	Left, 2 oz.	—	—	—
11	M.	5 months	1 oz.	Pneumococci and staphylococci	Yes	Right	—	+	Pneumococcal meningitis
12	M.	1	2 oz.	Pneumococci	Yes	Left, 10 oz.	—	Distant	—
13	F.	9 months	1 oz. turbid fluid	—	No	Right	—	—	—
14	M.	2	1 oz.	—	No	Right and left	—	—	—
15	M.	2½	4 oz.	Pneumococci	Yes	Right	No	—	Heart sounds inaudible; pericardium opened, drained; lived two months; died from measles; post mortem, sac of pus found shut off in posterior pericardium
16	M.	2½	2 oz.	Pneumococci	Yes	Right	+	C. sounds not heard	Admitted moribund
17	M.	1	1 oz.	—	Suspected	Left, 2 oz.	+	—	—
18	M.	1	1½ oz.	Pneumococci	No	Left	No	—	—
19	M.	6 months	1 oz.	<i>Bacillus influenzae</i> ?	No	Right	No	—	Meningitis
20	F.	16 months	3½ oz.	—	Suspected	Right and left	No	No; H.A.B. not felt or seen; sounds clear	—

THIRTY-FIVE CASES ASSOCIATED WITH EMPYEMA (continued).

No.	Sex	Age in years	Amount of pus in pericardium	Bacteriology	If diagnosed	Side of empyema	Cardiac dullness	Friction	
21	F.	22 months	2½ oz.	Pneumococci	Suspected, but rejected	Right	No	C. sounds clear	—
22	F.	4	10 oz.	Pneumococci	As left apical empyema	Right, very small	No displacement of heart, and sounds sharp and clear; dullness upwards and to left taken for empyema, apparently not explored?		
23	M.	1	3 to 4 oz. sides and back	Pneumococci	No	Left	Tu berculous caseating lungs		
24	M.	7	3 oz.	—	No	Right and left	—	—	—
25	M.	4	2 oz. "in pockets"	Pneumococci	Yes	Left	Pericardium opened, turbid fluid found; died two days later; pockets not opened; no signs; no + cardiac dullness; no friction or distant heart sounds; in creasing dyspnea without cough		
26	F.	3	5 oz.	Streptococci; no pneumococci	Suspected	Left, 12 oz.	H.A.B. fifth space not visible; no cardiac dullness to right; sounds distant, no friction		
27	F.	2½	1 oz.	Sterile?	No	Right	—	—	—
28	F.	1½	About 3 oz.	Pneumococci	No	Right and left "full"	No	Normal	—
29	M.	1½	"Many oz."	Pneumococci	No	Right and left	No	Normal	—
30	F.	9 months	About 3 oz.	Pneumococci	No	Left	—	—	—
31	F.	22 months	6 oz.	Pneumococci	Yes	Right	Yes, to right	Distant	—
32	M.	2½	1 oz.	Sterile	No	Right	Died suddenly whilst right empyema was being operated upon; caseating general tuberculosis found post-mortem		
33	M.	7 months	1 to 2 oz.	Pneumococci	No	Right and left	—	—	—
34	M.	4	2 oz.	Pneumococci, streptococci and <i>Bacillus coli communis</i>	No	Left, 10 oz.	Died an hour after empyema had been opened; very fetid pus; no intestinal lesion found; leucocytosis, 23,000		
35	F.	2½	4 oz.	Pneumococci	No	Right	—	—	Leucocytosis, 30,000

University College Hospital.

By F. J. POYNTON, M.D., and S. A. OWEN, M.B.

TABLE I.

Year	No. of cases per year	Age	Sex	Case book reference	No. of case
1894	0	—	—	—	—
1895	1	74	M.	M. 552, p. 682	1
1896	0	—	—	—	—
1897	2	{ 6 43	{ F. M.	{ S. 160, p. 465 M. 575, p. 614	{ 2 3
1898	1	5 mos.	F.	M. 590, p. 81	4
1899	0	—	—	—	—
1900	2	{ 7 12	{ M. F.	{ S. 179, p. 206 M. 616, p. 511	{ 5 6
1901	2	{ 17 mos. 66	{ M. F.	{ M. 622, p. 208 M. 624, p. 264	{ 7 8
1902	2	{ 18 51	{ M. M.	{ M. 631, p. 257 M. 639, p. 267	{ 9 10
1903	1	60	M.	M. 640, p. 65	11
1904	1	41	M.	M. 647, p. 304	12
1905	1	7	F.	M. 656, p. 298	13
1906	3	{ 28 7 52	{ M. F. F.	{ M. 670, p. 379 M. 675, p. 257 M. 669, p. 195	{ 14 15 16
1907	1	9	M.	M. 683, p. 491	17
1908	1	18	M.	M. 689, p. 548	18
1909	3	{ 4 30 7	{ M. M. F.	{ M. 2316/09/m M. 422/09/R M. 3140/09/SL	{ 19 20 21

Totals, 1894-1909, 0 to 3—total, 21 = 13 males and 8 females ; cases, 1 to 21.

TABLE II.—APPROXIMATE DURATION OF ILLNESS FROM COMMENCEMENT TO DEATH OR DISCHARGE.

No. of case	Result	Post-mortem
1 ... 10 days	Death	Yes
2 ... 7 days	"	"
3 ... 14 days	"	"
4 ... 3 days	"	"
5 ... 17 months	Recovery from operation on pericardium; death subsequently from cerebral abscess; post-mortem	
6 ... 15 days	Death	Yes
7 ... 15 days	"	"
8 ... 7 weeks	"	"

TABLE II (continued).

No. of case			Result	Post-mortem
9	...	6 days	Death	Yes
10	...	8 weeks	"	"
11	...	2 days	"	"
12	...	10 days	"	"
13	...	3½ weeks	"	"
14	...	1 week	"	"
15	...	3½ weeks	"	"
16	...	5 days	"	"
17	...	6 days	"	"
18	...	10 months	"	"
19	...	6 weeks	"	"
20	...	9 days	"	"
21	...	2 months	Recovery	Operation

Summary—

Maximum duration	...	17 months
Minimum duration	...	2 days
Deaths and post-mortems	...	20
Operations on pericardium	...	2
Recovery	...	1

TABLE III. (a)—PREVIOUS ILLNESSES WHEN STATED.

No. of case	
6	Rheumatism, three previous attacks
7	Measles two weeks prior to onset
8	None
9	Measles, chicken-pox, whooping-cough, influenza; C ₂ H ₂ O + lately
10	Winter cough, three attacks gout
12	Rheumatic fever
13	Croup, measles two years ago
14	None
15	Rheumatism two years before
16	Winter bronchitis
17	Scarlet fever, measles, influenza
18	Scarlet fever two years ago
19	Bronchitis since birth
20	Measles
21	Measles, rheumatism, chicken-pox, winter cough, diarrhoea

TABLE III. (b)—OCCUPATIONS (WHERE STATED).

No. of case		Sex
8	Caretaker	F.
9	Glazier	M.
10	Coachman	M.
11	Smith	M.
14	Tobacconist	M.
18	Railway porter	M.
20	Packer	M.

TABLE IV.—DIAGNOSES.

No. of case		
1	...	Pneumonia, pericarditis
2	...	Acute necrosis, pericarditis
3	...	Pneumonia, pericarditis
4	...	Acute miliary tuberculosis, pericarditis
5	...	Acute necrosis, purulent pericarditis, post-operative adherent pericardium, multiple and cerebral abscess
6	...	Rheumatic carditis
7	...	Broncho-pneumonia, pericarditis, empyema, meningitis
8	...	Granular kidney, pericardial effusion, pulmonary infarction
9	...	Pyæmia, pericarditis
10	...	Pneumonia, pericarditis, empyema
11	...	Pneumonia, pericarditis, empyema (double)
12	...	Pneumonia, pericarditis
13	...	Endocarditis, pericarditis, empyema, meningitis
14	...	Pneumonia, pericarditis
15	...	Septicæmia, pericarditis, endocarditis, empyema
16	...	Pleurisy, pericarditis
17	...	Acute necrosis, pericarditis, pyæmia
18	...	Chronic mediastinitis, chronic pericardial effusion
19	...	Chronic pneumonia, pneumococcic pericarditis and peritonitis
20	...	Pneumococcic pericarditis, pleural effusion, toxæmia
21	...	Pleurisy with effusion (? tuberculous)

TABLE V.—ANALYSIS OF INDIVIDUAL SYMPTOMS, SIGNS, &c.

- (a) No details given with regard to the following symptoms and physical signs and examinations:—
- (1) Præcordial œdema
 - (2) Friction fremitus
 - (3) Fluctuation
 - (4) Influence of change of position on the area of cardiac dullness
 - (5) Blood examination for bacteria
- (b) Præcordial bulging occurred in—
- (1) No. 6 (but in this case there was antecedent morbus cordis)
 - (2) No. 18 (case of chronic mediastinitis with 1½ pints fluid post mortem),
- (c) Dyspnœa occurred in 16—viz., Nos. 1, 3, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 18, 19 and 20
- (d) Cyanosis occurred in 14—viz., Nos. 1, 3, 6, 7, 9, 10, 11, 12, 13, 15, 16, 17, 18 and 20
- (e) Orthopnœa occurred in 7—viz., Nos. 1, 3, 6, 8, 10, 16 and 18
- (f) Œdema (local or general) occurred in 5—viz., 8, 10, 13, 18 and 21
- (g) Cough occurred in 9—viz., 3, 5, 7, 8, 14, 18, 19, 20 and 21
- (h) Pain occurred in 15—viz., Nos. 3, 5, 6, 7, 8, 10, 12, 13, 14, 16, 17, 18, 19, 20 and 21
- (i) Albuminuria occurred in 6—viz., 6, 8, 10, 13, 15 and 18
- Note.*—No. 8 was a case of granular kidney.
- (k) Hæmaturia occurred in 2—viz., Nos. 18 and 19.
- (l) Convulsions at onset in 1—viz., No. 4.
- (m) Delirium occurred in 5—viz., Nos. 2, 9, 15, 17 and 19
- (n) Rigors at onset occurred in 3—viz., Nos. 3, 12 and 16
- (o) Herpes occurred in 2—viz., Nos. 8 and 14
- (p) Albuminuric retinitis occurred in No. 10; post-mortem, right kidney granular, heart renal
- (q) Optic neuritis occurred in 1—viz., No. 5; post-mortem, cerebral abscesses

TABLE VI.

Examination by X-rays was done in one case only—viz., No. 21. X-ray examination confirmed clinical examination

Blood count in one case—viz., No. 18 (chronic mediastinitis and chronic pleural effusion) ; patient admitted April 22, 1908 ; blood examination (November 20) showed :—

Total red cells	4,700,000
Hæmoglobin	98 per cent.
C. index	0·9
Total white cells	15,000
Polymorphonuclears	81 per cent.

Examination of pleural exudates in same case (No. 18) :—

November 20—	Red corpuscles	10,000 per cmm.
	White corpuscles	140 per cmm.
	Some endothelial cells.			
	Lymphocytes, large	36 per cent.
	Lymphocytes, small	55 per cent.
	Polymorphonuclears	5 per cent.
	Hyaline	2 per cent.
	Transitional	2 per cent.
June 9—	Many red cells.			
	Total cell count	290 per cmm.
	Large lymphocytes	5 per cent.
	Small lymphocytes	80 per cent.
	Neutrophiles	15 per cent.
	Albumin	8 per cent.

Culture shows pneumococci.

Blood examination for Widal's reaction was done in case No. 20. Widal positive 1-30 only.

TABLE VII.

No.	Amount and nature of fluid in pericardium	Bacteriology	Cytology
1	$\frac{1}{2}$ pint pus (post-mortem)	No mention	No mention
2	4 oz. pus (post-mortem)	"	"
3	8 oz. sero-fibrinous fluid (post-mortem)	"	"
4	2 oz. pus (post-mortem)	"	"
5	? amount pus (operation)	"	"
6	15 oz. straw-coloured fluid (post-mortem)	"	"
7	? amount, distended with yellow watery pus (post-mortem)	"	"

TABLE VII (continued).

No.	Amount and nature of fluid in pericardium	Bacteriology	Cytology
8	10 oz. simple effusion (post-mortem)	No mention	No mention
9	? amount pus, pericardium transversely dilated (post-mortem)	"	"
10	1 pint pus (post-mortem)	"	"
11	? amount pericardium distended with turbid fluid (post-mortem)	"	"
12	6 oz. pus (post-mortem)	"	"
13	$\frac{1}{2}$ pint free fluid (post-mortem)	"	"
14	1 pint yellow pus (post-mortem)	"	"
15	10 oz. pus, turbid brown (post-mortem)	Diplo-streptococci	"
16	10 oz. creamy pus (post-mortem)	No mention	"
17	10 oz. blood-stained pus (post-mortem)	"	"
18	1 $\frac{1}{2}$ pint clear fluid (post-mortem)	No organisms	No. of cells, 108 per cmm.; lymphocytes, 81 per cent.; polymorphonuclears (neutro), 3 per cent.; endothelial cells, 6 per cent.
19	Pericardium full of liquid pus (post-mortem)	Typical diplococci of pneumonia	No mention
20	Large quantity, nearly clear, but contains much thick lymph (post-mortem)	Smear from lymph shows large number of encapsulated lanceolate diplococci and some short encapsulated chains up to streptococci	"
21	At operation pericardium thickened, large quantity of slightly turbid fluid was removed; tubercle, opsonic index, 0.47	From fluid removed at operation: (a) Cultures in broth (2), milk and agar-agar (incubated 48 hours), <i>nil</i> ; (b) subculture, <i>nil</i> ; (c) cultures from portion of pericardium: Broth and agar-agar (incubated 60 hours), <i>nil</i> ; (d) injection into guinea-pig, negative	There is a small quantity of blood mixed with the effusion. There are no flakes of lymph, but fluid coagulated spontaneously

TABLE VIII.

Two cases operated upon, Nos. 5 and 21. Diagnosis No. 5: Acute necrosis, purulent pericarditis; child recovered from this operation and died subsequently some months after from multiple cerebral abscesses. Diagnosis No. 21: Chronic pericardial effusion (? tuberculous). Details of the operation in these two cases will be found among the detailed case reports. The second case, No. 21, was discharged cured.

St. Bartholomew's Hospital.

By W. LANGDON BROWN, M.D., and A. E. GOW, M.S.

FATAL CASES.

Year	Under 5		5 to 10		10 to 15		15 to 20		20 to 25		25 to 30		30 to 35		35 to 40		40 to 45		45 to 50		50 to 55		55 to 60		Over 60		Total		No. of post-mortems during year
	M. F.		M. F.		M. F.		M. F.		M. F.		M. F.		M. F.		M. F.		M. F.		M. F.		M. F.		M. F.		M. F.		Total		
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.			
1894 ¹	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	164 ¹
1895	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	359
1896	2	1	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	4	6	362
1897	—	—	2	—	1	—	1	2	—	1	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	9	1	324
1898	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	1	302
1899	1	2	2	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	4	304
1900	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	2	320
1901	4	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	9	4	308
1902	3	1	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	5	5	312
1903	2	2	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	6	7	306
1904	2	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	10	5	328
1905	1	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	8	3	319
1906	2	2	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	6	4	326
1907	4	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	12	1	322
1908	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	10	1	329
1909 ¹	2	4	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	5	5	187 ¹
Totals	29	20	10	9	4	2	8	3	8	3	7	3	9	2	3	—	6	3	3	2	4	—	2	2	2	—	95	49	4892
																													144 ²

¹ Half year. ² = 2.94 per cent. of total post-mortems.

HEART.—PERICARDIAL.

	No. of cases	Less than 5 oz.	5-10 oz.	11-15 oz.	16-20 oz.	40 oz.	50 oz.	Not stated
Adhesions	11	—	—	—	—	—	—	—
Clear fluid	39	10	6	5	—	1	1	16
Sero-purulent	45	16	8	2	1	—	—	18
Purulent	39	16	2	—	2	1	—	18
Hæmorrhagic	20	3	5	1	1	1	1	8
Opalescent	1	—	1	—	—	—	—	—
Air	2	—	—	—	—	—	—	—

HEART—MYOCARDIAL.

Myocarditis	7
Infarction	1
Abscess in myocardium	1
Dilatation	(Right	6)	32
			Left	2)	
			General	24)	
Hypertrophy	(Right side	1)	26
			Left ventricle	12)	
			General	13)	

HEART.—ENDOCARDIAL.

Endocarditis	(Old	31)	70
			Recent	29)	
			Infective	10)	

Organisms found in pericardial fluid.

Typhoid bacilli	7
Pneumococci	30
Streptococci	(Longus	1)	10
			Pyogenes	1)	
			Not stated	8)	
<i>Staphylococcus pyogenes aureus</i>	2
<i>Bacillus coli communis</i>	1

PULMONARY.

Abscesses	2
Bronchitis	1
Bronchiectasis	1
Broncho-pneumonia	23
Brown induration	2
Collapse	32
Emphysema	2
Infarcts	6
New growth	3
Œdema	15
Pneumonia	(Right	7)	23
			Left	9)	
			Double	5)	
			Not stated	2)	
Tuberculosis	(Ulcerative phthisis	9)	13
			Miliary tuberculosis	2)	
			Tuberculous broncho-pneumonia	2)	

PLEURAL.						
Pleurisy, dry	16
Pleurisy, with effusion	Right	2	23
		Left	5	
		Double	8	
		Not stated	8	
Pleurisy, with purulent effusion	...	Right	12	34
		Left	9	
		Double	12	
		Not stated	1	
Pleural adhesions	13
Pneumothorax	1
Pyopneumothorax	2
Hæmothorax	4
Hydrothorax	13

MEDIASTINUM.						
Acute mediastinitis	3
Indurative mediastinitis	1
Caseous glands	2
Inflamed thymus	1
New growth	3

LIVER.						
Cirrhosis	5
Fatty	6
Hæmorrhages into	1
Nutmeg	39
Pale or cloudy	24
Perihepatitis	3
Tubercles in	4

SPLEEN.						
Cardiac	14
Enlarged	4
Infarction	7
Perisplenitis	9
Tubercles in	5

PERITONEUM.						
Ascites	15
Acute peritonitis	2
Acute suppurating peritonitis	7
Tuberculous peritonitis	1

KIDNEYS.						
Cardiac	22
Fatty	2
Infarction	8
Hæmorrhage into	1
Abscesses in	1
Nephritis	...	Sub-acute	1	22
		Chronic interstitial	15	
		Chronic parenchymatous	4	
		Chronic mixed	2	
Pale or cloudy	11
Tuberculosis	3

Medical Section.

April 26, 1910.

Dr. J. MITCHELL BRUCE, President of the Section, in the Chair.

Hospital Infection of Tuberculosis as exemplified by the Records of the Resident Staff of the Mount Vernon Hospital for Consumption and Diseases of the Chest for the past Fifteen Years.

By J. EDWARD SQUIRE, C.B., M.D.

A RECENT contribution [2] on the infection of consumption by Dr. Theodore Williams raises again the question of hospital infection, and refers to the original figures from the Brompton Hospital on this subject, which have been so widely quoted as evidence of the comparative immunity from tuberculous infection of the resident medical and nursing staffs of special chest hospitals.

These figures, and others which have been brought forward, appear, however, to be compiled from the results of inquiries into the health of the former residents and nurses rather than from any actual medical examination of the individuals concerned, and are thus inconclusive. In one case—that of Victoria Park, given by Dr. Andrew in his Lumleian Lectures [1]—the information appears to have been simply obtained from the secretary of the hospital.

As a further contribution to this question, I propose to give some figures from the Mount Vernon Hospital, which have at least the advantage of being compiled from actual examination of the individuals. As Dr. Williams's latest article gives particulars of the resident medical officers, I have added to my statistics particulars of the resident medical

officers obtained, like those of Dr. Williams, by inquiry only. It will, I think, be seen that the risk of infection in hospital is not entirely a negligible quantity, though the risk is a small one and affects the nurses and servants of the institution rather than the members of the resident medical staff.

The figures I am able to give cannot be taken to show every case of possible infection from hospital work, since they only refer to evidences of infection which were apparent up to the time when the individual left the hospital; it is, of course, possible that other cases may have developed tuberculosis after leaving.

The original report of the Brompton Hospital staff was presented to the British Medical Association by Dr. Theodore Williams at the annual meeting in 1882 [3]. As it covered a period of thirty-six years, the number of individuals who had been in residence must have been considerable, though it was perhaps impossible to give the actual numbers.

The table on p. 139 will show the nature of the material at present available for judging of the risk of hospital infection in this country.

These figures furnish some curious results, for we find that the dispensers, who come least in contact with the patients, show the highest proportion of consumptives (9.61 per cent.), whilst the nurses and female servants who are most continuously in the wards furnish the smallest proportion of consumptives (0.98 per cent.). This does not include those who were in residence at Brompton before 1867, for the numbers are not given in the report.

The resident medical officers (3.76 per cent.) and porters (4.18 per cent.) come between the dispensers and the female staff with a somewhat close approximation in the proportion of consumptives.

The Brompton report of 1882 has been very widely quoted as evidence that infection does not occur in properly conducted hospitals for consumptives.¹ It may be that other hospitals have felt some diffidence in publishing any cases of apparent infection of members of the staff which might lay the institution open to the presumption of insufficient attention to precautionary details.²

¹ Thus, in the first book which I took at random from my shelves in which reference is made to the matter, I read: "Although Brompton Hospital, London, has treated more than 15,000 cases of tuberculosis during the past twenty years, yet neither a nurse, a physician, nor an attendant has become infected."—"The Diagnosis and Treatment of Pulmonary Tuberculosis," by F. M. Pottinger. Ballière, Tindall and Cox, 1908.

² Dr. F. R. Walters, says: "No instance of infection has ever been reported from any modern chest hospital with even elementary precautions about the expectoration."—"The Sanatorium Treatment of Pulmonary Tuberculosis," 1909, p. 34.

TABLE I.

Hospital	Resident medical officers		Dispensers		Nursing staff		Female servants		Porters	
	Total	Became phthisical	Total	Became phthisical	Total	Became phthisical	Total	Became phthisical	Total	Became phthisical
Brompton, 1846-1867 ...	154	8 ¹	22	4	?	4 ¹	?	1	20	0
" 1867-1892 ...			107		32	1	0			
" 1892-1909, including Frimley ...	201	5 ²	23	0	—	—	—	—	137	7 ³
Victoria Park (Dr. Andrew 1884 ...	12	1	7	1	259 ⁴	1	—	—	34	1
Ventnor (quoted by Dr. Bulstrode, 1906) ...	—	—	—	—	208	6 ⁵	407	2	—	—
Crooksbury Sanatorium (quoted by Dr. Bulstrode, 1906) ...	5 ⁶	0	—	—	26 ⁶	0	76	1	—	—
Totals ...	372	14	52	5	600 + ?	12	515 + ?	4	191	8

¹ Only one resident medical officer and one nurse contracted phthisis whilst at Brompton.

² One contracted phthisis from a post-mortem wound. The remaining four became consumptive after leaving Brompton.

³ Five of these porters had to deal with the patients' clothing and bedding; another one was a pathological assistant, who became consumptive after an accidental wound.

⁴ Nurses and female servants are here included together. Thirty-three, in addition to the one referred to as phthisical, left because of illness, and of these "some, but certainly not all, were phthisical."

⁵ Three of these nurses appear to have had tuberculosis before going to Ventnor.

⁶ One resident medical officer and four nurses had been phthisical before they came to Crooksbury.

The Brompton report does not, it is true, show complete immunity from tuberculosis amongst those who had been in residence, though only one resident medical officer out of 150 is said to have contracted the disease whilst in residence, and only one or perhaps two individuals out of the many nurses and maidservants who resided in the institution during the thirty-six years with which the report deals. Nevertheless, it shows a surprisingly small incidence amongst those who have worked in the hospital as compared with the known prevalence of the disease amongst young adults in the general population of this country.

Dr. Williams, in his most recent report, seems to recognize a possibility of infection in general hospitals, since he states that, though amongst the house physicians none were affected during residence at Brompton, two subsequently died of acute phthisis when holding resident appointments in hospitals where cases of advanced consumption are admitted.

It is, I think, certain that residence in a well-regulated hospital or sanatorium for consumptives involves no great risk of infection; on the other hand, there are certain duties incidental to the care of consumptives which involve some risk, and therefore necessitate special precautions—such duties, for example, as cleansing sputum cups and flasks, handling soiled handkerchiefs, clothing, or bedding, and the performance of autopsies. Risks of direct infection from patients coughing—droplet or spray infection from sputum—or from general air infection from dried sputum should be practically non-existent in the wards of a well-ordered hospital.

Such as they are, the risks will therefore not be common to all the residents in the institution, but will vary with the special duties of the different members of the staff.

The visiting physicians may be left out of consideration; they are not living amongst the consumptives, and there should be little or no risk in examining the patients.

The out-patient physicians probably run some risk, since they examine a large number of patients at each visit, most of whom have not been instructed in the precautions necessary to minimize any possible danger to others.

The resident medical officers are in less close and less constant relation to the patients than the nurses, and their risk, if we exclude the performance of autopsies and possibly some danger in laryngoscopic examinations, is also slight.

The nurses are the most exposed to infection, and if there is any risk we should expect to find evidence of it in the incidence of consumption amongst the nursing staff. It is to be regretted that in his most recent communication in reference to the Brompton Hospital staff, Dr. Williams has left the nurses out of consideration. Wardmaids, scrubbers, laundry-maids and porters have special risks from dried sputum which make precaution in their work necessary; these individuals also do not readily appreciate the need for attention to detail, and soon neglect precautions if supervision is relaxed. We might expect that a certain percentage of residents, nurses, and servants would become consumptive, without therefore concluding that they became infected whilst engaged in the institution, and in consequence of their work there.

In order to assess the special risk attending work in a hospital for consumptives, we must endeavour to show that the incidence of the disease is proportionately greater amongst those engaged in such institutions than it is amongst those fulfilling similar duties in other hospitals, or else be able to trace the source of infection in individual cases, and show at least a probability of a definite relation between their special work and the contracting of the disease. The first condition—that is, the statistical comparison between the staffs of general hospitals and those of the special hospitals in relation to the incidence of consumption—is hardly possible; not only are the data difficult to obtain, but a very large proportion of the medical and nursing staffs of the special hospitals have been previously engaged in general hospitals, or pass on to the latter. A comparison might easily be made between the incidence of consumption amongst those who have been engaged in the special hospitals and amongst the general adult population, but this would be of little value unless the comparison were restricted to individuals of similar social grade and comparable kinds of employment. Even in respect of cases of consumption occurring in individuals engaged in special hospitals, whose duties expose them to definite risks, it is necessary to show that they were free from disease when they commenced their duties before we can reasonably infer that they became infected through their work. The statistics from Mount Vernon have a certain value from this point of view, since in the case of the nurses and servants, every individual was thoroughly examined on entry, and, in the great majority of those who were accepted, again on leaving. It is the rule at the Mount Vernon Hospital that every member of the nursing staff, every maidservant, and every man or boy employed in or about the hospital (engineers, porters, &c.) shall be examined before

being definitely engaged, and again on leaving. The chest is carefully examined, under the same conditions as obtain in the examination of the patients, by one of the visiting physicians detailed for this duty. This has been my duty for the past fifteen years, and from the records which have been kept, the following notes and statistics have been compiled. As I also see all members of the staff who are reported to be out of health, there should be little chance of their developing consumption without it being detected. Nurses naturally shrink from a thorough examination of the chest by residents with whom they are daily in communication; thus, if they find themselves failing in health, and suspect that their lungs may have become affected, they may keep the matter to themselves, and leave the hospital rather than report sick. Compulsory examination on entry by one of the visiting staff, besides ensuring the physical fitness of new members of the resident staff, paves the way for similar examination by the physician on the lightest hint of danger. I have several times been asked to examine nurses or servants merely because they or their friends had got a little nervous about possible infection. The origin of this practice at Mount Vernon is not without significance. A nurse (L. E. D.) came to the hospital from a provincial consumption hospital, and it was noticed that she had a hacking cough. She was persuaded to have her chest examined, and I found active mischief in the lungs, from which she died some six months later. According to her own account she had been off duty for an attack of acute bronchitis, and before she had completely recovered she was put on special duty to look after an advanced case until his death. It seems probable that, being in a condition of special susceptibility on account of her illness, she became infected from this patient. To avoid a similar occurrence in the future I suggested the systematic examination of all nurses and servants on entering on their duties and on leaving, which has now been carried out for over fifteen years. The numbers of which I have kept records comprise: From the Hampstead Hospital, 167 sisters and nurses, 92 maidservants, and 6 men and boys; and for the Northwood Hospital, 68 sisters and nurses, 73 maidservants, and 14 men and boys—a total of 420 persons. A certain proportion of these were rejected on the medical examination, others did not remain more than a few weeks, and some left without notice or were dismissed summarily for breaches of discipline or misconduct, so that the numbers examined on leaving will be considerably fewer than those mentioned above. It will be seen that evidences of past or present tuberculosis of the lungs were detected by medical examination in a not inconsiderable

proportion of the applicants for posts as nurses or maidservants, the condition being frequently unsuspected by the individual herself.

In addition to the above, there have been 33 resident medical officers, 3 dispensers, and a porter and caretaker at the out-patient department about whom particulars are available.

Such an inquiry as this may well be divided into three parts.

(1) The proportion of those desiring to enter the service of the hospital who have, or have previously had, some tuberculous manifestations.

(2) The evidence pointing to tuberculous infection whilst in residence in the hospital and the extent of the risk.

(3) The evidence of subsequent development of tuberculosis after leaving the hospital.

(I) EVIDENCE OF PREVIOUS TUBERCULOSIS IN APPLICANTS FOR APPOINTMENTS.

For the purposes of this inquiry we may take all the applicants who were examined on entry as to their physical fitness. Some of these were rejected at once as unfit for the duties required of them, others were allowed to remain though not physically sound, and were kept under observation. These were informed of the condition of the lungs, and were told that they would probably improve under the hygienic conditions observed in the hospital, and given the option of remaining. Most of them improved very considerably, and remained well; one or two did not improve, and left after a few months. It is noteworthy that of those who were found to have signs of tuberculosis, a large proportion were quite unaware of any such mischief, even when the signs showed active lesions. Others were aware that they had tuberculosis—quiescent or arrested—although they generally withheld the information on this point until it had been detected on examination.

The returns for Hampstead extend over fifteen years, those for Northwood only cover a period of five years.

The individuals examined on entry are comprised in the following table:—

		Hampstead		Northwood		Total
Matrons, sisters and nurses	...	167	...	68	...	235
Female servants	...	92	...	73	...	165
Men and boys	...	6	...	14	...	20
Total individuals	...	265	...	155	...	420

Amongst these there was evidence on entry of previous infection with tubercle as follows:—

	Hampstead	Northwood	Total
In nursing staff	25	7	32
In female servants	6	7	13
In men and boys	0	0	0
Total individuals	31	14	45

This gives a proportion of nearly 14 per cent. of the nurses and 8 per cent. of the maids who had been infected with tuberculosis before they applied for duty at Mount Vernon. There were in addition a few nurses and maids whose lung signs were not entirely satisfactory, and suggested a possible slight attack of tuberculosis; but, as the evidence in this direction was by no means conclusive, I have not included them as tuberculous in these returns. Of the forty-five individuals who gave evidence of previous infection, eight nurses and one maid had scars in the neck from old strumous glands, and one maid had an enlarged cervical gland which appeared to be actively tuberculous; three nurses and four maids had signs of old pleurisy, and of these, two nurses and one maid gave a history of repeated attacks—three in each case. Of the remaining twenty-eight individuals, the majority had signs pointing to arrested or healed mischief in the apex of the lungs, whilst in ten the signs pointed to still active tuberculosis in the lungs. One nurse had signs in the lungs as well as a scar in the neck.

Of those who, as the result of examination, were considered to have had tuberculous infection of the lungs, some were unaware of anything having been wrong; even those with active mischief—as, for example, the case (M. T.) detailed below—seemed to have no suspicion that their lungs were affected. Two or three were known to have been in hospital with pulmonary tuberculosis at some previous date, and some others as the result of inquiries eventually confessed to having been under treatment for consumption.

Some of the nurses and servants included in the above returns as previously infected were rejected as unfit, two were admitted as in-patients instead of being accepted for duty. Only the nurse (L. E. D.) referred to previously is known to have died of the disease, though one maid is still in the hospital as a patient.

Most of those who were taken on duty improved considerably during their residence. Four developed active mischief later, while doing duty

in the hospital. It will be noted that 13·6 per cent. of the applicants for appointment on the nursing staff presented evidence of tuberculosis, recent or remote, whilst of the servants applying only 7·8 per cent. showed signs of previous infection. This marked difference demands investigation, especially as tuberculosis is essentially a disease of the poor, and would be more prevalent in the class to which the servants belong than in that from which the majority of the nurses are drawn. Age might possibly have some influence, for some of the maids were younger than any of the nurses, and had not reached the age at which pulmonary tuberculosis is most common. The difference might be to some extent explicable on the supposition that the affected nurses might have selected such a hospital because the conditions of life were specially favourable to their condition, whereas the maids, coming from a more ignorant and prejudiced class, would avoid a consumption hospital if they had any suspicion that their lungs were not sound. In a few of the cases the nurses acknowledged that they had applied for posts at Mount Vernon because they thought it would be a good place for them as they had had their lungs affected, and it is generally known that a certain proportion of both medical officers and nurses in the various sanatoriums for consumptives have previously been patients at similar institutions. It is, however, impossible to overlook the possibility that previous occupation may afford the explanation of the greater proportion of nurses, as compared with servants, who have become affected with tuberculosis. Leaving out the cases of pleurisy and of strumous glands, we have 10 per cent. of the nurses and only 4 per cent. of the servants giving evidence of tuberculous infection. It seems possible that the nurses having been engaged in attending the sick and residing in hospital—at least during their training—may have become infected from their patients or have had their resisting power lessened by the effects of hospital work and residence. Many of the “nurses” were, however, probationers with no experience of nursing and no previous hospital residence, and, on the other hand, some of the maids had been employed in hospitals.

Of the tuberculous individuals, 17 (16 nurses and 1 maid) had had previous hospital residence, being nearly 37 per cent. of all who showed evidences of tuberculous infection. Of the tuberculous nurses alone, 48·5 per cent. had been in hospital.

The probable influence of hospital residence may be seen by the following comparison (p. 146).

(A) PROPORTION OF THOSE WHO HAD PREVIOUSLY RESIDED IN HOSPITAL WHO SHOWED SIGNS OF TUBERCULOSIS (EXCLUDING CASES OF STRUMOUS GLANDS).

Nurses	16 out of 70	=	22·75 per cent.
Maids	1 „ 17	=	5·28 „
Both	17 „ 87	=	19·54 „

(B) PROPORTION OF THOSE WHO HAD NEVER BEEN IN RESIDENCE IN HOSPITAL WHO SHOWED SIGNS OF TUBERCULOSIS (EXCLUDING GLANDS).

Nurses	17 out of 166	=	10·24 per cent.
Maids	12 „ 147	=	8·16 „
Both	29 „ 313	=	9·26 „

Whether the influence of hospital residence, as shown above, was in the way of direct infection from tuberculous patients, or was due to the effect on the general health rendering the individual more susceptible to infection from any source, or in causing a latent tuberculosis (due to infection before entering the hospital) to become active, it is, of course, impossible to determine. In any case it seems that the influence of general hospital residence cannot be altogether disregarded, since the proportion of tuberculous cases in those who had previously resided in hospital was twice as great as that amongst those who had never worked in hospital.

(II) THE EVIDENCE POINTING TO TUBERCULOUS INFECTION WHILST IN RESIDENCE AT MOUNT VERNON HOSPITAL.

The nursing staff of a hospital would seem to be most exposed to any risk there might be of direct infection through the air (by droplet infection, or from the general infection of the air of the wards from dried sputum) since their duties bring them into close personal relation with the patients for many hours a day. They probably have also to collect the soiled handkerchiefs, the used sputum cups and flasks, and to make the beds of the more advanced or febrile patients. Their risk would probably equal that of the near relatives and household of a consumptive patient who is confined to the house, except that the regulations of a well-disciplined institution insure attention to prophylactic detail which may be neglected in the average household. The maids are not so continuously in the wards, or so closely in attendance on the patients, and their risk should be much less; but they might be expected to be less careful in carrying out precautionary regulations. Some of the

men have special risks in connexion with the destruction of sputa, the disinfection of clothing and bedding, or in the post-mortem room.

If we were to rely upon the account which could be obtained from the individuals themselves we should only have to report a single nurse at Hampstead as having become consumptive whilst in residence, and possibly two at Northwood who developed tuberculosis—one in the hand and the other in the foot, though these latter may not have been aware of the nature of the mischief. If, however, we look at the results of examination, as shown in the following table, we see reason to doubt whether such a report would furnish a complete record of those who developed tuberculosis during their residence. Several of those recorded in this table had no definite symptoms of ill-health, and the majority had no suspicion that they had become infected with tuberculosis.

It will be seen on reference to the table (pp. 148-9) that, excluding doubtful cases,¹ fourteen members of the nursing staff, five maidservants, and two men developed tuberculosis during their residence in the hospital. Of those five nurses, three maids, and one man had been found on entry to have signs indicating previous infection. These cannot, therefore, be properly included in estimating the proportion of residents who may have become infected in the hospital. One of the maids who developed tuberculosis, and who ultimately died of the disease, contracted the infection apart from her duties. (*See case R. H. detailed below.*)

In calculating the percentage of cases of possible infection, only those members of the staff who remained in residence for three months or longer are included in the total of the residents, except in two cases who developed tuberculosis within three months of commencing their duties.

In making a critical examination of the cases referred to above as having developed tuberculosis during residence, we find that of the Hampstead staff five of the nurses may be considered to have developed tuberculosis in the lungs without any reasonable doubt, and of these two were recognized as tuberculous on entry. In three others the signs suggested pulmonary infection, but were not absolutely conclusive. In the remaining case (W. J.) if the swelling on the ankle was due to

¹ Most of the cases which I have called doubtful, both in the list of those who showed evidence of tuberculosis on entering the service of the hospital, and in that of the individuals who developed tuberculosis whilst in residence, had more definite physical signs of changes in the lungs—catarrh, consolidation, or induration—than many patients who are admitted into the wards as cases of pulmonary tuberculosis and sometimes proved to be tuberculous. These “doubtful” cases have not been included as tuberculous in any of the calculations of percentages.

TABLE II.—TABLE OF CASES DEVELOPING SIGNS OF TUBERCULOSIS (DEFINITE OR SUSPICIOUS) DURING RESIDENCE IN MOUNT VERNON HOSPITAL, 1894 TO 1909 INCLUSIVE.

No.	Occupation	Initial	Age	Date of Entry	When signs were noted	Condition noted on examination	Remarks
<i>Hampstead</i>							
1	Probationer-nurse	E. R.	17	April, 1895	Aug., 1897	Pleurisy left base	Developed cough six months later.
2	Nurse ...	M. T.	27	?	Sept., 1894	Creaking râles left back	Signs disappeared in about a month; doubtful.
3	Probationer-nurse	G. A.	19	Oct., 1899	Sept., 1900	Deficient resonance and interrupted breath sounds at right apex; had lost weight two or three months	Right apex showed quiescent mischief on entry.
4	Nurse ...	M. M.	25	Feb., 1900	Nov., 1900	Harsh bronchial breathing and crepitations right apex; few crepitations left apex	Now well (see report below).
5	Probationer-nurse	E. S.	19	Feb., 1903	April, 1905	Deficient resonance, high-pitched breath sounds, prolonged expiration right apex; harsh breathing left apex; temperature 100.2° F.	Left apex showed quiescent mischief on entry; had had pleurisy.
6	Sister ...	J. G.	31	Aug., 1904	July, 1905	Inconstant crepitations right lower lobe; much thinner	Left the hospital; believed to be well; doubtful.
7	Probationer-nurse	E. H.	23	March, 1905	Dec., 1905	Fine crepitations right front	Left the hospital; doubtful.
8	Probationer-nurse	E. D.	18	Feb., 1906	Oct., 1906	Bronchitic râles more marked on left; pleural friction left side	Seemed well on leaving the hospital, but her health broke down six months later.
9	Nurse ...	W. J.	20	May, 1906	Nov., 1906	Swelling of left ankle; no redness	Very bad family history of tuberculosis; ankle got quite well; doubtful.
10	Nurse ...	E. T.	23	April, 1907	Jan., 1908	Harsh breathing with prolonged expiration right apex	Possibly physiological; doubtful.
11	Wardmaid	R. H.	21	Oct., 1902	April, 1905	Cough a.m., creaking râles left suprapinnous fossa; moist crepitations right apex; tubercle bacilli in sputum	Died 1908 (see report).
12	Between-maid	C. B.	14	Oct., 1902	April, 1905	Few crepitations right apex	Probably temporary catarrh; doubtful.

13	Wardmaid	...	J. M.	21	May, 1905	Aug., 1905	Crepitations below right clavicle and in right supraspinous fossa; disappear on coughing	A delicate girl.
14	Between-maid	...	S. A. B.	17	Sept., 1903	July, 1904	Deficient resonance right apex; few post-tussive crepitations suprascapular region; deficient resonance left apex; moist crepitations after cough; tubercle bacilli in sputum	Admitted to wards.
15	Maid	...	E. D.	18	Nov., 1893	Feb., 1895	Harsh breath sounds and fine crepitations left apex; temperature 99° F. to 100.2° F., p.m.	Father phthisical; remained till 1897 and kept well.
16	Maid	...	B. C.	18	?	Feb., 1896	Deficient resonance both apices; creaking râles right supraspinous fossa; swollen cervical glands left side	Father died of phthisis; the lung signs were due to old tuberculosis before entry.
17	<i>Northwood</i> Nurse	...	E. H.	32	Nov., 1905	Jan., 1906	Occasional crepitations below both clavicles	October, 1907, creaking râles both apices, constant on right; had tuberculous cervical glands, aged 15. (See report); still on duty in the hospital. (See report).
18	Probationer-nurse	...	J. W.	26	Dec., 1907	March, 1908	Tuberculous finger from splinter	
19	Probationer-nurse	...	W. E.	21	March, 1905	Oct., 1906	Tuberculous metatarsal joint	
20	Probationer-nurse	...	J. D.	19	May, 1907	Jan., 1908	Acute tonsillitis; pleurisy left side	Probably rheumatic; complete recovery; left April, 1908.
21	Nurse	...	G. M.	25	Jan., 1905	Jan., 1906	Deficient resonance left apex; crepitations in both supraspinous fossae	Had pleurisy and pneumonia two years before entry.
22	Housemaid	...	L. M.	16	Nov., 1908	Jan., 1909	Tenderness and swelling in palm of hand	(See report); lungs showed old mischief on entry.
23	Housemaid	...	R. S.	16	Aug., 1906	May, 1907	Deficient resonance, prolonged expiration, and some crepitations above left clavicle	Left August, 1908; still keeps well; doubtful.
24	<i>Men</i> O.P. porter and caretaker (Fitzroy Square)	...	H. J.	40	Oct., 1906	Nov., 1907	Old mischief both apices; active in left lung	Died in the hospital; was invalided from army for hæmoptysis.
25	Engineer (Hampstead)...	...	W. C.	29	Aug., 1906	May, 1908	Fine crepitations at left apex; tubercle bacilli in sputum	Still on duty; now well (see report).
26	Page (Hampstead)	...	E. M.	18	Oct., 1908	Oct., 1909	Catarrhal condition right apex	Still on duty; doubtful.

tuberculosis it must be considered rather as the development of a latent infection than as an infection occurring whilst in residence, the activity being possibly due to a lowering of the resisting power by hospital work and residence—the influence of the hospital residence is in this case, at the most, indirect. There are thus only three cases of probable infection amongst the nurses during the past fifteen years which may be attributed to their work and residence at Hampstead.

Of the Northwood staff, two nurses developed signs in the lungs which pointed to tuberculous infection. One of these had suffered from strumous glands at the age of 15, and it is therefore conceivable that she had latent tuberculosis on entry; if so, the period of latency extended over seventeen years. The case of pleurisy cannot, in my opinion, be considered as an instance of tuberculous infection except on the supposition that all pleurisies are tuberculous. The pleurisy was preceded by an attack of tonsillitis which suggests a possible rheumatic origin. The case of tuberculosis of the finger is interesting, as it seems almost certainly to have been the result of local infection from a splinter of wood from the ward table which she ran into the finger. The case of tuberculosis of the metatarsal joint is less clear, as it is difficult to see how infection occurred, unless it were through the finger which she crushed in the door some weeks previously. (*See case W. E. detailed below.*)

Excluding those who were rejected as unfit on the admission examination, or for other reasons did not remain over three months, we have 155 nurses and 62 maids at Hampstead, and 60 nurses and 52 maids at Northwood.¹

We have, then, the following proportion of cases of probable infection:—

		Total		Developed tuberculosis		Excluding previous tuberculosis
Hampstead—						
Nurses	...	155	...	5, or 3·22 per cent.	...	3, or 1·93 per cent.
Maids	...	62	...	4, or 6·42 ,,	...	2, or 3·22 ,,
Both	...	217	...	9, or 4·14 ,,	...	5, or 3·20 ,,
Northwood—						
Nurses	...	60	...	4, or 6·6 ,,	...	2, or 3·3 ,,
Maids	...	52	...	1, or 1·92 ,,	...	1, or 1·92 ,,
Both	...	112	...	5, or 4·46 ,,	...	3, or 2·67 ,,
Both institutions together—						
Nurses	...	215	...	9, or 4·18 ,,	...	5, or 2·32 ,,
Maids	...	114	...	5, or 4·38 ,,	...	3, or 2·63 ,,
Both	...	329	...	14, or 4·25 ,,	...	8, or 2·43 ,,

¹ The comparatively large number of maids at Northwood is due to the fact that the laundry for both hospitals is at the Northwood branch.

Taking Table I for comparison, it will be seen that, excluding the Brompton returns before 1867 where the total number of nurses and servants is not given, we have—

Nurses	...	600, of whom 8 became consumptive	= 1·3 per cent.	} 0·98 per cent.
Maids	...	515, ,, 3 ,, ,, ,,	= 0·58 ,,	

It will, I think be evident, that the return of those who became infected with tuberculosis is obviously incomplete, as might be expected when the figures are obtained by inquiry only.

It may be well to point out here that the hospital at Hampstead is an older building than that at Northwood, and that all kinds of chest cases, including some quite advanced cases of consumption, are admitted to the wards, whilst the much newer hospital at Northwood is for early cases of pulmonary tuberculosis, and may be described as a model of the palatial type of sanatorium for consumptives.

Of the maids at Hampstead, six developed signs in the lungs suggesting tuberculous infection. In two of these the disease became acute, and one of them (R.H.) died. In the fatal case it will be noted (*see* case below) that there is sufficiently clear evidence that infection was contracted outside the hospital. In one other case the lungs showed signs of old mischief on entry. Two of the other three cases are open to doubt, and I have failed to trace the girls since they left the hospital. At Northwood two maids appear to have become infected. One of these—L.M., recorded below—is interesting as a case of probable local infection through a cut. She had signs on entry of previous infection of the lung. The other case gave doubtful signs in one lung. She remained at her work, and, though she has now left the hospital, I have ascertained that she keeps quite well.

Of the men, only two have showed any signs of mischief in the lungs. One of these was a caretaker at the offices in Fitzroy Square and out-patient porter there. He developed active tuberculosis, and ultimately died in the hospital; he had been invalided out of the Army for hæmoptysis. The other case was the engineer at Hampstead, whose duties included the destruction by burning of the contents of the sputum cups and flasks. He was never obliged to go off duty, though he remained under my care for six months. He is now perfectly well, and is still on duty at the hospital. It may be mentioned that all the men employed in the hospital are periodically examined every three months.

It is hardly possible to form any definite conclusion as to the

influence, if any, of hereditary predisposition since the numbers of those individuals in whom reliable information on this point is available is too small to be of any value. So far, however, as the figures give any indication, it would appear that the evidence points to the conclusion that it is undesirable that those who have a family history of tuberculosis should take work as nurses or maids in a special hospital for consumption. Of the Northwood staff, in whom the family history has been recorded, it appears that out of 71 nurses and 80 maids¹—a total of 151—of whom 22 gave a history of tuberculosis in the family, 9 nurses and 8 maids showed signs of previous tuberculosis on entry, and 3 others developed tuberculosis in residence. Thus we have 20 cases of tuberculosis in 151 individuals, or 13·24 per cent. Of these 20 cases of tuberculosis, 5 gave a history of tuberculosis in the family (25 per cent.), and 15 had no family history of tuberculosis (75 per cent.). Putting the figures in another way, we have 129 women with no family history of tuberculosis, and of these, 15 developed tuberculosis before or during residence—a proportion of 11·6 per cent. Of 22 women with a tuberculous family history, 5, or 22·7 per cent., developed tuberculosis. Thus the incidence of tuberculosis was considerably greater amongst those who belonged to tuberculous families. The numbers are, however, too small to be of any value.

The information as to family predisposition is less complete in the case of the Hampstead staff.

The average stay in the hospital is about the same for both nurses and servants—namely, about eighteen months; this is, of course, excluding those who did not stay as much as three months. Individual nurses and maids remained in the hospital several years, one as much as ten years and several for five years and over. It should be mentioned that a large proportion of the nurses are probationers who only come for one year, passing the remaining two years of their training in a general hospital.

(III) DEVELOPMENT OF TUBERCULOSIS SINCE LEAVING THE HOSPITAL.

I have not made any systematic attempt to estimate the proportionate number of nurses and maids who have developed tuberculosis since they left the Mount Vernon Hospital. In the first place it has been found

¹ Including some nurses and maids who were transferred to Hampstead, and are counted in the Hampstead figures in the other statistics in this paper.

almost impossible to get into communication with a large proportion of the former nurses and maids, the maids especially being most difficult to trace after a few years. In the second place such information as can be obtained is not altogether reliable, and cannot be corroborated by personal examination. I have obtained information regarding a considerable number of past nurses, and amongst those there has not been a single case of tuberculosis reported.

I have been the less persistent in seeking information since I have preferred to restrict this report as far as possible to facts ascertained by personal physical examination.

RESIDENT MEDICAL OFFICERS.

Although I have at different times examined several of the resident medical officers at their request, there has not been the same systematic examination of these residents as of the nursing staff and servants. Nevertheless, since the other reports which I have quoted for comparison give particulars of the resident medical officers, I give similar particulars.

Out of thirty-three past residents to whom I have written, twenty-seven have replied, one is known to be dead. Of the twenty-seven from whom I have recently had communications, all but four are able to state that they have never suffered from tuberculosis either before, during, or since their residence at Hampstead or Northwood. They generally agree that their residence did them good rather than harm, and several of them speak of putting on considerable weight whilst in the hospital.

Of the four exceptions, two had suffered from pulmonary tuberculosis before they came into residence, the disease having been arrested. Both of these remain well and at work.

One other pricked his finger at a post-mortem examination which led to a local infection of the hand. After about two years the hand became quite well. He is now, some twenty years later, still in good health. The remaining one developed several so-called tuberculous warts on the hands during residence nearly twenty years ago. Most of them have been cut out, and all are perfectly well; his general health has remained good. He writes that he quite recently reacted strongly to Calmette's test, but is in perfect health.

The dispensers, the secretarial staff, and the visiting medical and surgical staff furnish no cases of tuberculosis.

It would appear from the experience of the Mount Vernon staff that the risk of infection with tuberculosis in a special chest hospital is very small, and is apparently little, if at all, greater than in a general hospital. It is, perhaps, doubtful if there is any greater risk to the workers in any well-ordered hospital than amongst the general mass of town dwellers of similar social position. Infection with tuberculosis does occasionally occur as a result of the duties of a nurse, but probably not more frequently amongst the nurses in a hospital for consumptives than amongst the general body of nurses. Every occupation has its special risks, and the possibility of infection is one which members of the medical and nursing profession accept as a matter of course. The risk of tuberculous infection, even in a hospital for consumptives, appears to constitute a very slight danger.

ILLUSTRATIVE CASES.

Unsuspected Tuberculosis of Lung Detected by Examination on Entry.

M. T., aged 21, was examined on entry as wardmaid in November, 1908. She had been wardmaid at the Great Yarmouth Hospital. She was found to have active and advanced tuberculosis at the apex of the left lung entirely unsuspected. She was admitted to the wards as a patient. There was impaired resonance with prolonged expiration at the right apex. Diminished resonance, prolonged expiration, and moist crepitations over the greater part of the upper lobe and at the apex of the lower lobe on the left side. Tubercle bacilli were found in the sputum. She remained in the hospital (Hampstead and Northwood) until October, 1909, when she left much improved. She has not been at work since, but wrote at Christmas that she felt well and was anxious to get a situation as soon as possible.

Apparent Infection during Residence in Hospital.

Probationer-nurse M. M., aged 25. Examined on entry, February, 1900, and found healthy. In November, 1900, she was reported to me, having got thinner and lost weight. She was easily tired, slept badly, and suffered from headaches. I found the breath sounds harsh at the right apex, where there were some inconstant crepitant râles. A week later crepitations were detected at both apices, and she was put off duty. A month later the signs were more definite, especially at the right apex. She remained under my care for about five years before I could certify that the mischief was completely arrested. During this time she spent three and six months respectively at sanatoriums, the second occasion being in consequence of activity in the left lung,

the right being quiescent. For the last three years—July, 1902, to July, 1905—she was on night duty at the hospital. During the whole period she never had any expectoration. She has, since leaving the hospital, held posts at private sanatoriums, and remains well, reporting herself to me for examination at intervals when in London. Nurse M. was engaged for some months before her breakdown in charge of the out-patient waiting room, and it seems possible that she became infected from the out-patients, who are not sufficiently careful in the matter of expectoration until they have been instructed in the necessary precautions. The out-patient department at Hampstead has since been closed.

R. H., aged 21, wardmaid. Entered the hospital in October, 1902, and was then quite healthy. In April, 1905, she was "run down," had some morning cough, and, on examination, was found to have harsh breathing and some moist crepitations in the apex of the right lung. Examination of the sputum showed the presence of tubercle bacilli. She was taken off duty and put under treatment. She remained under treatment at Hampstead and Northwood until her death in 1908. It was found that this girl had become engaged to a patient who had tuberculous laryngitis, and who, when he left the hospital, took rooms at Hampstead. There were thus many opportunities of direct infection.

J. W., aged 26, probationer-nurse. Entered December, 1907. Healthy. In February, 1908, she ran a splinter into the left first finger whilst rubbing a ward table. A week later the finger was swollen and brawny, and Mr. Berry laid the track of the splinter open in the Royal Free Hospital, where the condition was pronounced to be tuberculous. In October, 1908, the finger was practically well, but the nurse was very anæmic and not very strong. After a short time she improved considerably, and is now still on duty in the hospital.

W. E., aged 21, probationer-nurse. Entered March, 1905. She was anæmic, the weight was subnormal; she had a somewhat flattened chest, but the lungs were healthy. In the summer of 1906 she crushed one of her fingers in a door, and shortly after the finger showed signs of local suppuration. She went for a holiday to Switzerland, and soon after her return she was found to have lost 3 lb. in weight since her holiday; she was anæmic, but there were no abnormal physical signs in the chest. In October, two months later, she complained of pain in the foot when walking. Mr. Berry examined the foot, which was slightly discoloured and swollen on the dorsum, and diagnosed tuberculosis of the metatarsal joint. She left the hospital a month later, and is now—October, 1909—perfectly healthy and well.

L. M., aged 16, housemaid. On entry in November, 1908, she was noted as tall and slight, with slight lateral curvature of the spine. The chest was healthy, except for a few creaking sounds on the left front. In January, 1909, she cut her hand with a knife. A few days later she broke some crockery, and thinks she got something into the wound of the previous cut. A week later there was swelling in the palm of the hand, which was tender on deep pressure. She was admitted into the Royal Free Hospital under Mr. Berry.

W. C., aged 29, engineer. Entered 1906. Was quite well until May, 1908, when he complained of pains in the chest and general weakness. On examination the only abnormal signs heard in the chest were a few rhonchi scattered throughout the lungs. He quickly improved, and felt well again, but about a month later he felt pain in the sternum and commenced to cough. No history of chest disease in the family. There was prolonged expiration at the right apex with a few crepitant râles. Rhonchi all over the back. At the left apex crepitations were heard both back and front. Tubercle bacilli were found in the sputa. A week later he had some pleurisy (dry) at the right base, and the physical signs at the right apex were more marked. By October 1 the chest was practically clear again, and he has since remained perfectly well. He is still on duty at the hospital.

REFERENCES.

- [1] ANDREW, J. Lumleian Lectures, Royal College of Physicians, *Lancet*, 1884, i, p. 837.
- [2] WILLIAMS, C. THEODORE. *Brit. Med. Journ.*, 1909, ii, p. 433.
- [3] *Idem. Ibid.*, 1882, ii, p. 618.

DISCUSSION.

Dr. THEODORE WILLIAMS said that when he read the abstract of Dr. Squire's paper he was under the impression that it was a description of what had taken place at Mount Vernon Hospital during the last fifteen years ; he did not think it likely that the original statistics which he and Dr. Cotton published concerning the Brompton Hospital would be discussed, but only mentioned ; nor did he think some of the data, or the method of obtaining them, would be questioned. But one feature of Dr. Squire's paper was that he dwelt particularly on the fact of his (Dr. Williams's) data not being complete, because there was hearsay evidence with regard to the medical officers and nurses. But this was not correct. It was true that medical officers were not required to undergo a medical examination, but no nurse was admitted to Brompton Hospital who had not undergone medical examination. The same was the case with the porters, and, he believed, with the servants also. When a nurse came into residence she was from that time under the supervision of the resident medical officer, who had to look after her health throughout her residence. So it was not a matter of hearsay ; it was just as much careful inspection as Dr. Squire himself gave. That was especially the case in the first set of statistics, for it would be remembered by many old Brompton house-physicians that during that period there was a remarkable resident medical officer (Mr. Vertue Edwards), who married the matron, and both were in residence at the hospital about thirty years. They knew every nurse and their relatives and acquaintances ; and he (Dr. Williams) availed himself of the valuable evidence which Mr. Edwards had accumulated. Some criticisms had been made as to the effect of those nurses who were under observation for only a short time ; but they were generally under observation for a long time. They were generally kept in touch with by Mr. and Mrs. Edwards until their death. With regard to medical officers, he was glad to see present more than one old house physician of the Brompton Hospital ; old Brompton house-physicians had done so well that they were conspicuous figures in the medical profession, and there was no difficulty in tracing them. Dr. Squire had twice mentioned that he (Dr. Williams) stated that in some of the cases the disease was contracted in a general hospital. In his (Dr. Williams's) last paper he mentioned that among the house physicians there were two who died of acute tuberculosis. Two became medical officers at a poor-law infirmary and at a general hospital respectively, and both died of phthisis some months after leaving Brompton Hospital. The paper of Dr. Williams stated : " It is possible the disease may have been contracted at Brompton or at the hospital infirmary in which they died, in both of which institutions cases of advanced consumption are admitted." He did not wish to say that many such cases were contracted in general hospitals ; he did not enter into that question. He only mentioned that out of 181 cases there were two which ended fatally, and of those he had given

an account. Those were all the cases occurring since 1882, and they had been included in the statistics published in 1909. He had communicated to the profession two sets of statistics, all carried out in the same way, and showing the same excellent results, though the last set was more favourable than the first. He was very sorry about the Mount Vernon nurses; he did not clearly understand from Dr. Squire whether the cases which he examined and found to be tuberculous were admitted as nurses. If they were, they ought not to have been. The great feature about the lecture which he (Dr. Williams) published was not the amount of disease found among medical men or nurses—that was trivial—it was the amount occurring among porters and pathologists—i.e., among those who did the post mortems and assisted in purifying the sputum. Everything was done to protect them; they were supplied with india-rubber gloves, &c., and if they carried out the precautions there was no doubt they would be safe. But they did not always do so; they sometimes dissected without gloves, and thus contracted infection; or else they handled the sputum with the naked hands. Mr. Stanley Boyd had published two or three very striking cases in porters. With regard to the other cases, he went carefully into the matter last year, before preparing his lecture for Stockholm, intending to have a discussion at the International Association; but he was prevented by an accident from attending there, and the lecture was published afterwards in the *British Medical Journal*.¹ He still held that there was very little contagion, and Dr. Squire's conclusions were very much the same as his own, only Dr. Squire thought there were more cases among nurses. Dr. Squire did not give an account of all the other officers. In his (Dr. Williams's) he included almost everybody connected with the hospital, including laymen and the chaplain. He thanked Dr. Squire for his statistics, as they were valuable, and would add considerably to our knowledge on the subject and enable trustworthy conclusions to be arrived at.

Sir SHIRLEY MURPHY said he attached much value to the paper, not only because the observations were made by a skilled clinician, but were made by the same man working all the time; therefore the investigation was comparable from beginning to end. He thought that what was required was the necessity for similar observations upon a similar population not exposed to tuberculous infection in hospital. Until that was obtained, figures were being dealt with the actual meaning of which it was somewhat difficult to interpret. He would be glad to see somebody make similar observation in the examination of female elementary school teachers. Dr. Squire had made two points: first, he had dealt with the nurses who were previously employed in institutions and those who were not previously so employed, and he had stated that those who were previously employed in general hospitals suffered more frequently from tuberculosis than did those who were not previously employed. But Dr. Squire had not given the ages of those nurses who were employed. Possibly the question of age might determine the greater incidence upon the nurses previously

¹ *Brit. Med. Journ.*, 1909, ii, p. 433.

employed; they were possibly older people, and were approaching the age at which tuberculosis attacked in greater degree. He had with him London figures for seven years, showing phthisis death-rates at ages in the female sex. Their interest was in the fact that they showed that there was an increasing incidence of death from about 5 years up to the age-period from 35 to 45 years, and any interval of time—three or four years or more—would make a difference in the proportion of general population which would be found phthisical if they were examined by Dr. Squire. The eighteen months or two years that the nurses were in hospital might be expected to make an appreciable difference in the figures. Dr. Squire's figures showed that some 10 per cent. of the nurses and servants were involved when they entered the hospital and 12·5 per cent. when they left. Those figures might possibly be explained by the fact that the female staff had lived a few years longer. He attached great value to the paper, but it had not impressed him that Dr. Squire had afforded any evidence that those cases were produced because they were exposed to tuberculous infection in the hospital. On the bacteriological hypothesis, no doubt the increase should have been brought about in that way; but evidence of infection from person to person was always lacking when the subject was dealt with statistically.

Professor OSLER said the profession was greatly indebted to both Dr. Williams and Dr. Squire for their consoling investigations—so comforting to physicians and nurses. He had often remarked to relatives fearful about catching consumption that the place with least risk was a well-conducted sanatorium. He had no accurate figures, but his impression was that, in general hospitals, the medical students, doctors, and nurses were much more affected with tuberculosis than were the other members of the community. Figures collected some years ago (which he regretted he had not just now) showed a considerable incidence of the disease among medical students and nurses in general hospitals. From what was known about the widespread prevalence of tuberculosis, it was safe to say that all the Brompton and Mount Vernon house-physicians and nurses became infected—all were tuberculous, but very few consumptive, and the absence of active disease spoke well for the dietetic and general sanitary arrangements of both Brompton and Mount Vernon Hospitals. All who mixed much with tuberculous patients got infected, but remained well so long as they took care of themselves and kept the soil in a condition unfavourable for the growth of the seed.

Dr. HERON desired to add his praise of Dr. Squire's paper and the great care with which he had collated his facts. He (Dr. Heron) had never been impressed with the statistics regarding the infectiveness or otherwise of tuberculosis in British hospitals, because he was sure that of late years the conditions favourable to infection were much more strongly marked in the homes of the poor than in hospitals. It was from the poor that most of the cases of tuberculosis came. That disease was becoming from year to year much rarer among the well-to-do sections of the community. Some years ago his colleague

Dr. Chaplin and himself investigated the question of the infectivity of dust taken from likely places in Victoria Park Hospital. The result was published in the *Lancet* in 1894.¹ Only 2 out of 100 guinea-pigs used were infected as a result of injecting into them dust which was thought likely to be infected with tubercle; and those two were from the dust obtained from the central air-shaft of the hospital. Even the dust obtained from corners of the wards which were difficult to get at for cleaning did not yield infection. A well-conducted hospital he regarded as a less dangerous place than the homes of the poor. In the last twenty-one years, at Victoria Park Hospital, he had known of six cases of tuberculosis amongst the nurses. There were thirty-five to forty nurses, constantly engaged, and the hospital contained 164 beds, less than half of which were occupied by people suffering from consumption in one stage or another. It was the duty of one of the members of the visiting staff to examine every nurse before she was admitted to the service of the wards, and the slightest suspicion of tuberculosis insured her rejection. Since 1889 five of the resident medical staff had left the hospital with tuberculosis. One of them had tuberculosis before he took up his duty, though it was unknown to himself. Had there been the slightest suspicion that he was tuberculous he would not have been allowed to enter on his hospital service. But one should be careful in drawing conclusions from such an inquiry as the present, for it was not uncommon to find that non-medical people somehow arrived at the conclusion that the disease occurred in some way other than by infection. That was a most unfortunate fallacy to get abroad; for, notwithstanding the rarity of infection in hospitals, there were 55,000 or 56,000 people dying annually in England and Wales from tuberculosis, always a result of infection. That fact must be driven home, and then there would be greater efforts on the part of the people to help to exterminate this disease of tuberculosis, which could arise in no way but by infection.

Dr. NATHAN RAW said that Dr. Squire had brought forward a subject which was not only of intense importance to the medical profession, but to women who took up nursing as a profession, and also to the laity themselves. During the last twenty-two years he had made careful observations on the subject, and he agreed with one of the speakers that observations made by one man over a long period were more valuable than the compiled statistics of several. His figures almost agreed with those of Dr. Squire. During the last twelve years, out of 420 nurses and servants employed in the hospital with which he was connected, eight nurses developed pulmonary tuberculosis; and out of seventeen house surgeons two had symptoms of the disease. No sisters, charge-nurses, nor matrons developed it; those that did were practically all probationers. He could not be sure they had not tuberculosis which escaped his detection on examination before entrance to the hospital, but out of such a number a certain number would develop the disease according to the usual ratio. He believed nurses were most prone to develop tuberculosis

¹ *Lancet*, 1894, i, p. 14.

in asylums, secondly in general hospitals, and least of all in the open-air wards of a well-conducted sanatorium. During the last four years no nurse had developed tuberculosis. The risk of infection in nursing tuberculosis was less than in typhoid, and more nurses had developed typhoid. The discussion would perhaps allay the fear which probationers had of nursing tuberculous patients; recently some nurses had declined to nurse such cases. A very few years ago it was the practice of a Sanatorium Committee to advertise for a matron or nurse or resident medical officer and insert a clause, "Those who have had tuberculosis preferred." It showed the great change of feeling on the matter which had now come over the profession and public in the matter. He affirmed that no nurse or officer should take up work in nursing or attending tuberculous patients if they had the slightest tendency to the disease. He believed there was more tuberculosis among school teachers than among nurses, showing that schools which were not properly ventilated were a source of danger to the community.

Dr. WETHERED said he would speak from general experience rather than from statistics. One was more likely to contract the disease in a general hospital than in a special one; but he would add a word as to the unreasonable fear of the infectivity of tuberculosis among the public. Investigations had shown that there were more sources of infection than from dried sputa of tuberculous subjects—namely, from tuberculous milk or other food. The nature of the life and the bad ventilation, such as in schools, brought that latent infection into activity. With all the precautions in well-conducted hospitals and sanatoria, infection might be regarded as negligible. He was once consulted by a lady in great trepidation lest a sanatorium a mile away from her house should be a source of infection. If such discussions as the present would allay such a feeling, they would be very beneficial.

Dr. SQUIRE, in reply, said he was sorry if Dr. Williams thought he (Dr. Squire) had criticised his figures unduly. Dr. Williams had misunderstood part of his criticism. He did not wish to suggest that Mount Vernon was the only hospital where nurses were medically examined on admission; it was, he believed, the usual thing, and very necessary. What he had insisted on was the value of a routine examination of nurses and servants on leaving, especially when they had been working in a chest hospital. He did not intend to quote Dr. Williams as stating that any nurses got infected at a general hospital. He believed he (Dr. Squire) had come across a fact of considerable importance from the figures—namely, that the risk of becoming affected with tuberculosis was greater in a general hospital than in a special one, and he had credited Dr. Williams with having pointed out this possibility in his paper. With regard to the acceptance of nurses who had previously had tuberculosis, he did not accept nurses for duty who had active tuberculosis, but he did not reject those who had arrested tubercle. One medical officer was present who might have endorsed what he had said; he had been accepted, although he had suffered from tuberculosis, and now, after five or six years' residence in the hospital, he

was still alive and well. Sir Shirley Murphy had mentioned the question of age, and the figures which that gentleman gave were extremely interesting, because they showed the amount of difference which age made in the proportionate incidence of tuberculosis. The difference in the ages which might be supposed to separate the probationers from those who had been in residence in the hospital made, in the ordinary population, a difference of between nine and fifteen deaths per 1,000 living at the respective age-periods, whereas in the figures given in his paper the percentage incidence of tuberculosis between those who had not been in residence in hospital and those who had was as nine to nineteen, which seemed to show that age alone would not completely cover the disproportion. He, however, agreed that the point with reference to age was important and could not be disregarded. Professor Osler confirmed the impression which he had himself arrived at with regard to infection at a general hospital; but he touched only slightly on a thing which he (the speaker) would have been glad to hear emphasized—namely, the possibility that all, at least in large cities, were, in a sense, tuberculous. Evidence was accumulating which would lead one to say there was a close analogy between tuberculosis and pneumonia, in that just as people might have pneumococci inside them a long time without particular harm until something happened to make the soil fertile, so tubercle bacilli might lie latent in the body until the circumstances were favourable to their development and dissemination. The onset of tuberculosis was not necessarily coincident with the infection, but might depend on the accident of fertilization of the soil at the moment. If so, attention had to be given to general health rather than to dried sputum in formulating preventive measures. The fact that Dr. Nathan Raw's and his own figures were nearly alike tended to confirm the conclusions arrived at in his paper. He was much gratified that the paper had given rise to such an interesting discussion.

Medical Section.

May 24, 1910.

Dr. J. MITCHELL BRUCE, President of the Section, in the Chair.

A Consideration of the Results of the Pancreatic (Cammidge) Reaction in 1,475 Cases.

By P. J. CAMMIDGE, M.D.

IN a paper which I read before the Royal Medico-Chirurgical Society on March 13, 1906,¹ I described an improved method of performing the so-called "pancreatic" reaction in the urine,² and gave my experience of the reaction with 100 specimens derived from 94 cases. Since then I have considerably extended my experience, and now have records of over 1,500 samples of urine derived from 1,475 cases. These I propose to consider in the present communication. The large majority were sent to me by medical men in various parts of the country for diagnostic purposes, and it is owing to their kindness

¹ *Med.-Chir. Trans.*, Lond., 1906, lxxxix, p. 239.

² The details of the method are as follows: A specimen of the twenty-four hours' urine, or of the mixed morning and evening secretions, is filtered several times through the same filter paper and examined for albumin, sugar, bile, urobilin, and indican. A quantitative estimation of the chlorides, phosphates, and urea is also made, and the centrifugalized deposit from the urine examined microscopically for calcium-oxalate crystals. If the urine is found to be free from sugar and albumin, and of an acid reaction, 2 c.cm. of strong hydrochloric acid (specific gravity 1.16) are mixed with 30 c.cm. of the clear filtrate, and the mixture gently boiled on the sand-bath in a small flask, having a long-stemmed funnel in the neck to act as a condenser. After ten minutes' boiling the flask is well cooled in a stream of water, and the contents made up to 30 c.cm. with cold distilled water. The excess of acid present is neutralized by slowly adding 8 grm. of lead carbonate. After standing for a few minutes to allow of the completion of the reaction, the flask is again cooled in running water and the contents filtered through a well-moistened, close-grained filter paper until a perfectly clear filtrate is secured. The filtrate is then well shaken with 4 grm. of powdered tribasic lead

in furnishing me with the results of operations, and the after-histories of their cases, that I am able to compare my analytical findings with the clinical conditions present in most of them. I take this opportunity of thanking them for their courtesy and ready response to my letters of inquiry.

The cases may be divided into nine main groups :—

- (I) Acute or subacute pancreatitis, 17.
- (II) Chronic pancreatitis, 859.
- (III) Pancreatic calculi, 4.
- (IV) Pancreatic cysts, 4.
- (V) Pancreatic infantilism, 1.
- (VI) Cancer of the pancreas, 73.
- (VII) Miscellaneous, 467.
- (VIII) Normal, 50.
- (IX) Cases re-examined after operation, 55.

(I) ACUTE PANCREATITIS.

Thirteen cases in Group I gave a positive pancreatic reaction. Six of these were operated on, and the pancreas was found to be acutely inflamed in all, with disseminated fat necrosis in 5. The diagnosis made at operation was confirmed by post-mortem examination in 3. Gall-stones were discovered in the common bile-duct of 2. One had a history of repeated attacks of jaundice with spasm, and had probably suffered from common-duct cholelithiasis, but there was no jaundice at the time of the analysis. In three the pancreatitis was the sequel of an attack of mumps, but in four other cases of the same disease without abdominal symptoms, which I have also included in this group, the urine

acetate, and the resulting precipitate removed by filtration, as clear a filtrate as possible being obtained by repeating the filtration several times if necessary. The large amount of lead now in solution is removed either by treatment with a stream of sulphuretted hydrogen or by precipitating the lead as a sulphate. For the latter purpose the filtrate is well shaken with 2 grm. of finely powdered sodium sulphate, the mixture heated to the boiling point, then cooled to as low a temperature as possible in a stream of cold water, and the white precipitate removed by careful filtration; 10 c.cm. of the perfectly clear transparent filtrate is made up to 18 c.cm. with distilled water and added to 0.8 grm. of phenylhydrazin hydrochlorate, and 2 grm. of powdered sodium acetate, contained in a small flask fitted with a funnel condenser. The mixture is boiled on a sand-bath for ten minutes, and then filtered hot through a filter-paper moistened with hot water into a test-tube provided with a 15 c.cm. mark. Should the filtrate fail to reach the mark, it is made up to 15 c.cm. with hot distilled water. In well-marked cases of pancreatic inflammation a light-yellow, flocculent precipitate should form in a few hours, but it may be necessary to leave the preparation to stand overnight before a deposit occurs.

gave no reaction. Two cases gave a history of indigestion and abdominal discomfort, extending over several months previous to the attack. Three of the patients operated on died, and 3, including the 2 in which gall-stones were found, recovered. All the patients not operated on died, except the 3 in which the pancreatitis followed mumps. The analysis of the urine showed albumin in 6. No sugar was found in any of them, but 1 developed glycosuria a year after the onset of the acute symptoms. Acetone was present in 4, and 6 showed aceto-acetic acid. An excess of indican was found in 7, including the 3 cases of mumps. Both the cases with gall-stones, and 1 of those not operated on, contained bile, and these, as well as the 3 cases of mumps and 2 others, gave a well-marked reaction for urobilin. Calcium-oxalate crystals were found in the urinary deposits from 7. I have had the opportunity of examining specimens of urine obtained subsequent to operation from 4 of the cases, and found that the pancreatic reaction gradually diminished in intensity, but was still present to a slight extent one month later. One case showed the presence of acetone and aceto-acetic acid a fortnight after operation. The bile disappeared from the urine of both the cases of gall-stones, but urobilin was met with in one case for twenty-seven days afterwards, and in 2 others at the end of a week and ten days respectively.

(II) CHRONIC PANCREATITIS.

Chronic pancreatitis is rarely, if ever, a primary disease, but when once it is established it is liable to steadily progress, and may seriously interfere with the metabolic processes of the body, eventually leading to glycosuria in some cases. The earlier symptoms of the disease are usually so indefinite, and more or less completely masked by those of the primary condition giving rise to it, that it is generally only by an appeal to the methods of the laboratory that its presence can be definitely recognized. Even then it is necessary to exercise a good deal of caution, and to interpret the results of a careful and complete analysis of the urine and fæces, with, in some cases, the data obtained from a test meal, in the light of experience, and to check these by the clinical signs and symptoms. By this means chronic inflammations of the pancreas can usually be detected, and in many instances the amount of damage sustained by the gland can be surmised. To merely make a diagnosis of chronic pancreatitis is, however, not enough; the primary cause of the condition must also be determined

if a successful line of treatment is to be pursued, and by considering the clinical condition, together with the results of an analysis of the excreta, this can generally be done.

The causes of chronic pancreatitis are numerous, but from the clinical information supplied me concerning the cases I have examined, and the evidence obtained from analysis, I have been able to divide them into the following classes: (1) Those associated with gall-stones, malignant disease of the common bile-duct or ampulla of Vater; (2) those probably arising from diseases of the intestine, including duodenal and gastric ulcer; (3) those associated with diseases of the heart, lungs, or blood-vessels; (4) pancreatitis due to metastatic deposits of growth in the pancreas, from malignant disease in other organs. Additional causes that have been described by various authors include stenosis of the duodenal orifice of the common bile and pancreatic ducts from ulceration, &c.; obstruction of the ducts by hydatid membrane, &c.; tubercle, syphilis, influenza, and other zymotic diseases, and alcoholism, but of these I have no definite examples in my series of cases.

I have examined specimens of urine from 264 cases in which gall-stones were found at operation, and of these 149 gave a positive reaction and 115 a negative result. In 188 cases the gall-stones were in the common bile-duct, and of these 131 (69.6 per cent.) gave a positive and 57 (30.4 per cent.) a negative pancreatic reaction, percentages which, it will be noticed, correspond fairly closely with the proportions of cases in which, according to Helly, the common bile-duct lies embedded in the pancreas (62 per cent.), and runs in a groove on the posterior surface of the gland (38 per cent.). On dividing these cases into groups according to whether bile was or was not detected in the urine, it is found that out of 128 with jaundice, 91 (71 per cent.) gave a positive pancreatic reaction; and out of 60 non-jaundiced cases, 40 (66.6 per cent.) gave a positive result. Twelve of the former and 9 of the latter had sugar in the urine, varying from traces to 6.8 per cent. in the jaundiced, and to 7.8 per cent. in the non-jaundiced. Acetone was present in 6 of the cases with jaundice, including 2 with glycosuria; and in 2 in which the urine was free from bile pigment, both of which had glycosuria. Aceto-acetic acid was found in 17 jaundiced cases, 2 having glycosuria, and 14 of the non-jaundiced, 5 of which had sugar in the urine. Indicanuria was noticed in a larger proportion of the non-jaundiced than the jaundiced cases, an excess of indican being found in 31 of the former and 20 of the latter.

A more or less marked reaction for urobilin is a most useful indication of the probable presence of gall-stones in the common duct, especially when they are "floating." In this series of 188 cases it was met with in 123 (65·4 per cent.), being more common where the calculi did not give rise to jaundice—viz., 47 cases (78·3 per cent.)—than in those in which there was obstruction of the bile flow—viz., 76 cases (59·3 per cent.). A deposit of calcium-oxalate crystals, which, as I have pointed out, is a very constant accompaniment of chronic pancreatitis, was met with in 98 cases, or 69 per cent., of those in which there was evidence of pancreatitis. Nine of the cases in this group, which had given a positive pancreatic reaction during life, were examined post mortem, and the diagnosis of pancreatitis confirmed microscopically. Specimens of urine were obtained after operation in 24 cases, 20 having previously given a positive pancreatic reaction. In 14 the reaction was found to have disappeared, at intervals varying from one to four weeks after the operation, but in 6 it still persisted when the last analysis was made. Four of the jaundiced cases showed bile pigment in the urine, and 2 gave a reaction for urobilin. Two of the cases with glycosuria showed no trace of sugar, 1 having had 4·8 per cent. and the other 0·4 per cent. previous to operation, and in a third it fell from 7·8 per cent. to 3·7 per cent. after operation.

Thirolloix and Maugeret¹ have maintained that cholecystitis is a common, if not the commonest, cause of pancreatitis, the inflammation spreading by way of the lymphatics, from the inflamed gall-bladder to the head of the pancreas. My experience does not tend to confirm this, for out of 76 cases in which gall-stones were found in the gall-bladder or cystic duct at operation, only 18 gave a positive pancreatic reaction, and were stated to have pancreatitis. Fifty-eight gave a negative result; as did also 17 cases clinically diagnosed as cholecystitis, 6 of which were operated on, that I have included in the miscellaneous group. Sugar was not detected in the urines of any of these cases. Two had traces of acetone, and 13 gave a more or less marked reaction for aceto-acetic acid. Sixteen showed an excess of indican. Bile pigment was found in the urines of 11, including 9 of those that gave a positive pancreatic reaction. Fifteen gave a reaction for urobilin, and of these 10 gave a pancreatic reaction. Calcium-oxalate crystals were met with in the deposit from the urines of 18 of those that gave a positive pancreatic reaction, and in 4 of those with which no reaction was obtained.

¹ "Thèse de Paris," 1908.

A positive pancreatic reaction was given by the urines of 2 out of 8 cases of malignant disease of the common bile-duct, and 2 of growth at the ampulla of Vater. In the 2 former that gave a reaction the growth was said to be fused with the head of the pancreas, and had probably started in the pancreatic portion of the duct. Neither of the cases of malignant disease of the ampulla of Vater was examined after death, but it is probable that the pancreatitis was due, in part, to blocking of the bile and pancreatic ducts, and in part to invasion of the pancreas by the growth. The urine of 1 of the cases of malignant disease of the common duct contained 0·85 per cent. of sugar, and a trace of albumin. Only 1 of the 10 cases in this group gave a reaction for urobilin, and in this the liver was being invaded by the growth.

Chronic pancreatitis is, I believe, most commonly secondary to disease of the intestine. In most of these cases there is probably an infection of the pancreatic ducts by direct extension from the duodenum, the pancreatitis being primarily of the catarrhal type; but in others, such as typhoid fever, it is possible that the parenchyma of the gland may be primarily affected by organisms carried by the blood and lymph streams. If it is allowed that gall-stones are usually the result of an infection of the bile passages from the intestine, the pancreatitis met with in cholelithiasis would also fall into this class, at least in some instances, for it is not unlikely that the bile and pancreatic ducts are often simultaneously infected owing to their close anatomical relations. I have, however, considered cases in which gall-stones were found apart, and in the present group only include 403 cases in which the clinical symptoms and results of the analysis of the urine and fæces pointed to there being disease of the intestine other than a malignant growth. Of these 403, the urines of 211 gave a positive pancreatic reaction, and 192 a negative result.

In 194 a diagnosis of intestinal catarrh, or enteritis, colitis, appendicitis, dysentery, hyperchlorhydria, or indigestion was made, and of these 103 gave a positive reaction, including 52 cases diagnosed as intestinal catarrh, 23 of colitis, 3 of appendicitis, 13 of hyperchlorhydria, and 12 of indigestion. In the 91 giving a negative reaction were 25 cases of intestinal catarrh, 50 of colitis, 10 of appendicitis, 1 of dysentery, and 5 of indigestion.

The explanation of the presence of the pancreatic reaction in about two-thirds of the cases of intestinal catarrh or enteritis is, as I have explained, probably an inflammatory condition of the pancreas due to

an extension of the catarrh of the intestinal walls along the pancreatic ducts. A similar explanation also probably holds good for those cases of colitis and appendicitis in which a reaction was obtained, for in many instances these conditions are associated with a more or less marked pathological state of the entire length of the intestinal tract. A considerable number of the cases which I have included under the heading of intestinal catarrh had in addition some colitis, as an analysis of the fæces showed, but the disease was evidently not very marked clinically, as it was not mentioned in the reply I received to my letter of inquiry. Hyperchlorhydria, as I pointed out some time ago,¹ is a possible cause of pancreatitis. The constant excessive stimulation of the pancreas by the secretin formed by the excess of hydrochloric acid is likely to bring about abnormal physiological activity of the gland, to be followed by degenerative changes if the over-stimulation is long continued. Such I believe to be the explanation of the positive reaction with the 13 cases diagnosed as hyperchlorhydria. Indigestion is too vague a term to be of much importance in such an investigation as this, but in 12 out of the 17 cases in which that diagnosis was made the results of the pancreatic reaction suggested that the pancreas was undergoing active degenerative changes.

Albumin was found in the urines of 18 of these cases, acetone in 9, aceto-acetic acid in 37, an excess of indican in 125, urobilin in 60, and calcium-oxalate crystals in 62. The high proportion (64 per cent.) in which indicanuria was a symptom is interesting, for it has been claimed by some observers that a pathological excess of indican in the urine is an important sign of pancreatic disease, while others have stated that a marked diminution is the rule. My own experience is, however, that neither the one nor the other can be relied upon as a symptom of pancreatitis, but that a pathological excess indicates rather the condition of the intestine and suggests that any pancreatitis that may be present is probably the result of an ascending infection from the duodenum, while the absence of indicanuria points to some other cause. The association of urobilinuria with indicanuria and a positive pancreatic reaction, which occurred in 52 cases, indicates that the infection involved the bile as well as the pancreatic ducts. Twenty-seven of the cases in this group were operated upon, and in 25 the pancreas was said to be enlarged and harder than normal. A positive pancreatic reaction had been obtained with the urines of all. Seven

¹ "Intestinal Indigestion," *Lancet*, 1909, i, p. 223.

were examined post-mortem and pancreatitis found microscopically in 4 which had given a urinary reaction during life; the other 3 had given no reaction and the pancreas appeared normal. In 8 cases a second analysis of the urine was made at intervals varying from two weeks to two months after the first examination. Two still gave a positive reaction, but with 6 a negative result was obtained. Both those which gave a positive result still had marked indicanuria, and one showed traces of urobilin and bile. I have not included in this group a number of cases of glycosuria in which there was marked indicanuria, as I propose to deal with the subject of diabetes, and the application of the pancreatic reaction to the examination of urines from such cases, subsequently; but I may now state that I am convinced, from the observations I have made, that diabetes is a very common result of long-continued intestinal catarrh slowly inducing changes in the pancreas.

Catarrhal jaundice is generally assumed to be due to an inflammatory process in the duodenum extending into the ampulla of Vater, which is closed either by a swelling of the mucosa or by a thick plug of mucus. Virchow described such a case, and Osler, among others, has observed a similar condition. Eppinger¹ has more recently published a case in which it was proved after death that the occlusion of the duct was due to a hyperplastic condition of the lymphoid tissue of the mucosa of the duct following acute gastro-enteritis, and he suggests that some cases of catarrhal jaundice occurring in young people are to be ascribed to angina-like attacks of lymphoid swelling in persons of a "lymphoid" constitution. However this may be, it may be taken for granted that catarrhal jaundice is usually, if not always, associated with gastro-intestinal disorders, and that owing to the close relation of the bile and pancreatic ducts, the latter will probably suffer along with the former, so that there is likely to be some pancreatitis. Mayo Robson has suggested that in the more chronic cases the persistence of the jaundice is dependent upon compression of the common bile-duct by the swollen and inflamed head of the pancreas. A diagnosis of catarrhal jaundice was made in 53 of the cases I have examined, and in 42 of these a positive pancreatic reaction was obtained, suggesting that in rather over 79 per cent. the condition was associated with some active degenerative changes in the pancreas. The jaundice was accompanied by an excess of urobilin in the urine in 13 cases, and calcium-

¹ *Wien. klin. Wochenschr.*, 1909, xxi, p. 480.

oxalate crystals were found in the deposit from 26, all of which had given a positive pancreatic reaction. Fifteen cases were submitted to operation because of the persistence of the jaundice, and in all of these the pancreas was enlarged and harder than normal. A previous examination of the urine had pointed to the presence of pancreatitis. I had the opportunity of re-examining the urines from 8 cases, and obtained a positive pancreatic reaction with 2, both of which were still jaundiced.

Occasionally one meets with chronic pancreatitis due to direct extension of an inflammatory process from a neighbouring organ, such as the stomach or intestine, but more commonly the pancreatitis in gastric and duodenal ulcer appears to be due to the associated gastro-intestinal catarrh extending to the pancreatic ducts and setting up a catarrhal pancreatitis. I have examined specimens of urine from 50 cases of ulcer of the duodenum and 47 of gastric ulcer, all of which were confirmed by operation. Twenty-seven (54 per cent.) of the former and only 5 (10·6 per cent.) of the latter gave a pancreatic reaction. In 2 of the cases of gastric ulcer, and 1 of duodenal ulcer, the ulcer was said to be eroding the pancreas. In 20 out of 27 cases of duodenal ulcer there was indicanuria, and in 4 of the cases of gastric ulcer an excess of indican was also found, pointing to abnormal putrefactive changes in the contents of the upper part of the intestine, and suggesting that the pancreatitis was due to a catarrhal condition spreading from the duodenum to the pancreatic ducts. The comparatively high proportion of cases of duodenal ulcer in which a pancreatic reaction was obtained, and its rarity in almost exactly the same number of cases of gastric ulcer, lend support to this view. A second examination of the urine was made in one of the cases with adherent gastric ulcer three months after the first, and a positive pancreatic reaction was again obtained. The pancreas was examined post-mortem in 1 of the cases of adherent gastric ulcer and advanced cirrhotic changes were found spreading a considerable distance into the substance of the gland.

Two other diseases in which I have obtained evidence of pancreatitis, and in which it is probable that the condition is due to an ascending infection from the duodenum, are sprue and pernicious anæmia. The former is well recognized as a disease of the intestine of microbic origin, and many cases of so-called pernicious anæmia are, I believe, dependent upon an abnormal intestinal flora with auto-intoxication and blood destruction. Out of the 13 cases of sprue, 8 gave a positive

reaction, and in 2 of these post-mortem examination showed pancreatitis. Four out of the 6 cases diagnosed as pernicious anæmia gave a pancreatic reaction. In 1 of these, examination after death revealed chronic inflammation of the pancreas and an ulcerated condition of the duodenal mucous membrane. Operation on 2 also tended to confirm the diagnosis of chronic pancreatitis. It is noteworthy that all the cases of sprue and pernicious anæmia had marked indicanuria.

Chronic pancreatitis in tuberculous cases may arise from deposits of tubercle bacilli carried by the blood stream to the pancreas, but Carnot, Ancelet, Opie, and others have described chronic inflammation of the gland without any specific lesion, and Carnot has experimentally proved that such changes may follow the injection of tubercle bacilli and their toxins into the duct and parenchyma. I have not been able to obtain a post-mortem in either of the 2 cases of tuberculosis of the intestine that gave a positive pancreatic reaction, and so cannot say whether secondary deposits were present or not in the pancreas; but, as there was marked indicanuria in both, I am inclined to think that the pancreatitis may possibly have been the result of infection by way of the pancreatic ducts with bacteria originating in the intestine. Six other cases diagnosed as tuberculosis of the intestine gave a negative reaction, as did also 3 of pulmonary tuberculosis.

The pancreatitis that is occasionally met with as a sequel of typhoid fever is possibly due to a specific infection travelling up the ducts, and I have therefore included in this group 8 cases in which typhoid was present at the time of the examination or the history suggested that it was probably connected with the symptoms of which the patient complained. It is also possible, however, that typhoidal pancreatitis may arise from infection of the blood, which is always present. Six gave a positive pancreatic reaction, but this result cannot be taken as any indication of the proportion of cases of typhoid in which there is involvement of the pancreas, as both the negative cases were suffering from the disease at the time the examination was made, while the 6 that gave a positive reaction were convalescent or had a history of an attack and were sent to me because the symptoms suggested chronic pancreatitis. One such patient, who came from America, had glycosuria, and I succeeded in isolating organisms having the appearance and reactions of typhoid bacilli from his fæces. In this case there was also evidence of cholangitis and there was an excess of indican in the urine.¹

¹ "Pancreatic Glycosuria ten years after Typhoid Fever," *Lancet*, 1909, i, p. 1739.

Nearly all observers are agreed that the pancreas is frequently affected in cirrhosis of the liver. Klippel and Lefas have concluded, as the result of their observations, that the two are due to the same ætiological factor, but that the pancreatic condition is independent of, and not secondary to, the lesion in the liver. As alcohol is generally acknowledged to be a common exciting cause of cirrhosis of the liver, and its use to excess gives rise to disturbances of the gastro-intestinal tract, it is not a very far cry to assume the possible dependence of the associated inflammatory changes in the pancreas upon an ascending infection from the duodenum along the pancreatic ducts, at least in some cases. I have therefore included 21 cases in which there was said to be cirrhosis of the liver and pancreas in this group. Fourteen of these gave a positive pancreatic reaction, and 7 a negative result. In all of these the state of the liver and pancreas was determined at operation, and in one which had given a positive result a post-mortem examination was made. I may also add 7 cases of cirrhosis of the liver that gave no reaction, but were not examined on the operating table or in the post-mortem room. Six of the cases had more or less bile in the urine, and 4 showed an excess of urobilin. Indicanuria was present in 6, and calcium-oxalate crystals were found in the urines of 7. One had a slight glycosuria and gave a positive pancreatic reaction.

In the large majority of cases already mentioned the results of the pancreatic action were borne out by the clinical condition and observations made at operation or in the post-mortem room; but I now come to a group of 11 in which, although I am assured that the pancreas was enlarged and hard, no pancreatic reaction was obtained. These I have put down as cirrhosis of the pancreas, as I believe they were cases in which, although there was more or less cirrhosis, the result of past inflammation, no active degenerative changes were going on in the gland substance at the time the urine was examined. To these may be added the 5 cases with gall-stones in the common bile-duct in which no pancreatic action was obtained, but the pancreas was found to be enlarged and thickened at operation, and the 5 cases in which there was cirrhosis of the liver and pancreas, with a negative reaction mentioned above.

Basing his conclusions on the post-mortem records of Guy's Hospital, Hale White is of opinion that disturbances of the circulation are much commoner than other causes of "congestion, cirrhosis, and hardening of the pancreas," but in my experience circulatory disturbances are relatively uncommon causes of pancreatic troubles. Fleiner and Hoppe-

Seyler both state that arterial diseases cause nutritive changes in the parenchyma, which degenerates and is replaced by fibrous tissue. I have, however, only met with 10 cases giving a positive pancreatic reaction, in which it was stated that the pancreatitis which was suspected to be present was probably dependent upon arterio-sclerosis. Long-standing difficulties of venous flow, due to chronic disease of the heart, liver, or lungs, may cause fibrosis of the pancreas, as of other organs; but Opie is of opinion that passive congestion is an unimportant factor in the production of chronic pancreatitis. I have examined the urines from 6 cases in which chronic pancreatitis was said to be associated with heart disease, and obtained a positive reaction in all of them.

In common with other organs of the body the pancreas is liable to be the seat of metastatic deposits of growth from malignant disease occurring in other tissues, and it may also be invaded by growths situated in the stomach, duodenum, and neighbouring organs. The resulting inflammatory changes are usually local, but give rise to alterations in the urine, which can be recognized by the pancreatic reaction. I have examined the urine from 92 cases of malignant disease and obtained a positive pancreatic reaction in 34. These included 14 of cancer of the stomach, 12 of malignant disease of the intestine, 4 in which the primary growth was situated in the gall-bladder, 2 in the prostate, and 2 in the breast, both the last named being cases in a late stage, one of which had been examined earlier and had given a negative result. No pancreatic reaction was given by the urines from 58 cases in which the growth was situated in the tongue (2), eye (1), stomach (17), intestine (21), gall-bladder (2), kidney (3), urinary bladder (1), prostate (1), breast (7), and uterus (3). The diagnosis was confirmed in 27 cases by operation, and in 4 by post-mortem examination. In the remainder it was based upon the clinical signs and symptoms. Sugar was found in the urines of two cases. One of these is of particular interest, for it demonstrated very clearly the dependence of the pancreatic reaction on degenerative changes in the pancreas and its disappearance with the destruction of the gland, when advanced glycosuria had been established. The patient was first seen in December, 1906. There was then an abdominal tumour, which it was thought might be pancreatic, but an examination of the urine gave no pancreatic reaction and there was at that time no sugar. An exploratory operation was performed by Mr. Mayo Robson, and a growth of the first part of the duodenum found, but it was said to be quite free from the pancreas. On January 18 a second specimen of urine was examined

and found to be free from sugar, but to give a well-marked pancreatic reaction, suggesting that the pancreas was then involved in the disease. At the request of the patient's friends the abdomen was reopened a few days later, and it was then found that the growth had invaded the pancreas. In the early part of May examination of the urine showed 5.25 per cent. of sugar, and a modified pancreatic reaction gave a positive result. A month later the sugar had increased to 7 per cent., and a much less marked pancreatic reaction was obtained. In July the urine contained 7.25 per cent. of sugar and the pancreatic reaction gave only a few crystals. In August 7.5 per cent. of sugar was present, and no crystals were found on carrying out the modified pancreatic reaction. In October the urine contained 9.5 per cent. of sugar and the pancreatic reaction was negative. The patient died deeply jaundiced on November 5. Another case in this group of considerable pathological interest has been published by Mr. Mayo Robson and myself.¹ A post-mortem examination showed that the pancreatitis, which the urinary examination had indicated, was due to direct extension of a malignant growth of the gall-bladder along the common bile and pancreatic ducts.

In 63 cases the urine gave a positive pancreatic reaction; but I have not received any reply to my inquiries as to the final diagnosis and subsequent course of the case, and so cannot say whether there was clinical evidence of pancreatitis or not.

(III) PANCREATIC CALCULI.

Pancreatic calculi are exceeding rare. In 1903 Oser was only able to collect 70 recorded cases. The calculi probably arise as a consequence of morbid changes in the pancreatic secretion consequent upon inflammatory alterations in the glands and ducts.² It has been supposed that, like gall-stones, they are the result of infection of the ducts by micro-organisms. I have had the opportunity of examining the urine from 3 cases in which pancreatic calculi were discovered at operation, and one in which they were found post mortem. Three gave a positive pancreatic reaction, and one, in which there was a large amount of sugar and the whole pancreas appeared to be represented by fibrous tissue, gave a negative result. Calcium-oxalate crystals were discovered in the centrifugalized deposit from the urines of all four.

¹ *Lancet*, 1907, ii, p. 508.

² "The Pancreas: its Surgery and Pathology," 1903.

(IV) PANCREATIC CYSTS.

Chronic pancreatitis is present in many cases of simple cyst of the pancreas, and there is no doubt, both on experimental and clinical grounds, that it is a frequent, and probably the most common, cause of the condition.¹ Cysts are, however, only likely to occur in the later stages of the disease, when a considerable amount of fibrous tissue has formed. This, by its contraction, may cause constriction of the ducts in some places, while other parts are pulled upon and dilated; the pancreatic secretion will then tend to accumulate in the dilated portions and undergo chemical changes, by which its physical characters are altered and its absorption interfered with. It is unlikely, therefore, that a cyst of the pancreas will be associated with a positive pancreatic reaction unless active degenerative changes are going on in the gland, as the result of the pressure effects it may produce in the remainder of the organ, or the cause of the pancreatitis still persists. Of the 4 cases of pancreatic cyst that I have examined, the urines of 2 gave a positive pancreatic reaction, and in 2 it was negative. One of the latter had, however, been operated on and the cyst drained for some three months before I saw him, when he still had a fistula. I had the opportunity of examining the urine and fæces from one of the cases, giving a positive result three years after the first analysis. The urine then contained 4·5 per cent. of sugar, although it had been quite free before, and showed traces of aceto-acetic acid, but no acetone. A positive pancreatic reaction was still obtained, suggesting that the active degenerative changes associated with the presence of the cyst in the first instance were still progressing, and were likely to bring about still more marked glycosuria. In one case, which was diagnosed clinically as a pancreatic cyst, but the urine of which gave no pancreatic reaction, the tumour proved at operation to be a mass of tuberculous glands pushing forward the pancreas, and in another a hydatid cyst of the liver, which had been similarly diagnosed, caused no pancreatic reaction in the urine.

(V) PANCREATIC INFANTILISM.

I have examined the urine and fæces from one case diagnosed as pancreatic infantilism² and obtained a positive pancreatic reaction with the former. There were also traces of aceto-acetic acid and urobilin, but no acetone, bile-sugar, or albumin in the urine.

¹ *Ibid.*

² Byrom Bramwell, *Scot. Med. and Surg. Journ.*, 1904, xiv, p. 321.

(VI) MALIGNANT DISEASE OF THE PANCREAS.

The difficulty of successfully diagnosing cancer of the pancreas from chronic pancreatitis on the clinical data alone, especially when the latter is associated with obstruction of the common bile-duct, is now generally acknowledged to be very great. Even when the additional facts to be obtained by a chemical and microscopical examination of the urine, fæces, &c., are taken into account, a few cases present a somewhat puzzling combination of signs which it takes considerable experience to interpret satisfactorily; but in the large majority of instances the laboratory analyses furnish information which is more conclusive than can be obtained in any other way. In cases of primary malignant disease of the pancreas the urinary pancreatic reaction is usually negative, but in a certain percentage a positive result is obtained, owing probably to the inflammatory reaction set up in the adjacent gland tissue by the irritation of the growth and the blocking of the duct. Of the 73 cases diagnosed as cancer of the pancreas that I have examined, the urines of 24 (33 per cent.) gave a positive reaction, and 49 (67 per cent.) a negative reaction. The diagnosis was confirmed by operation in 18, and by post-mortem examination in 7. Two of the latter had given a positive result, and 5 a negative reaction, which corresponds with the proportions diagnosed clinically and at operation, which had given a positive and negative result respectively. Bile was present in the urines of 67, usually in large quantities, but only 5 gave a reaction for urobilin, which contrasts markedly with the cases in which the obstruction of the bile-duct was due to gall-stones. Acetone was found in 5, and acetoacetic acid in 18. Only 16 gave an abnormal indican reaction. Calcium-oxalate crystals were found in the deposits from 8, but 3 of these had histories of old gall-stone seizures, so that it was possible that there was interstitial pancreatitis as well. These 3 all gave a positive pancreatic reaction. Sugar was found in the urines of 6, but always in small amounts, varying from 0·4 to 0·9 per cent. Two of these cases gave a positive pancreatic reaction.

It will be seen from these results that, while the pancreatic reaction in the urine is of value as showing the presence or absence of pancreatitis in a case of persistent jaundice, it does not differentiate between those in which the inflammation is the result of malignant disease and those in which it is of simple origin. To attain this end the whole of the results secured by a complete qualitative and quantitative analysis

of the urine and fæces must be considered, and these again be checked by a consideration of the clinical signs and symptoms. A quantitative and qualitative analysis of the fæces is of the utmost importance in suspected cases of cancer of the pancreas. Personally, I never care to venture an opinion on the symptoms and urinary examination alone. The percentage of unabsorbed fat, the relation between the unsaponified and saponified fats, the presence of stercobilin and occult blood, and the results of the pancreatic insufficiency test, must all be determined and carefully considered, for from them one can determine with a considerable degree of accuracy the extent of the pancreatic mischief, also whether the biliary obstruction is quite complete or not, and from that infer whether one is dealing with a case of growth or simple inflammation of the pancreas.

(VII) MISCELLANEOUS.

In a miscellaneous group of 467 cases, 461 gave a negative pancreatic reaction. Many of these were sent to me because it was thought, from the clinical condition, that there was some inflammation of the pancreas, but subsequent observation tended to disprove this and support the opinion based upon the urinary analysis in about one-third of the cases. In the remaining two-thirds I have not received any reply to my letters of inquiry. Those in which I have been favoured with a final diagnosis include gastritis (16), dilated stomach (14), intestinal obstruction (3), tuberculous glands (2), pneumonia (1), phthisis (5), pleurisy (1), pleural effusion (1), adenoma of the thyroid (1), floating kidney (6), stone in the kidney (10), nephritis (9), tuberculosis of the kidney (3), cystitis (4), tuberculosis of the bladder (1), myoma of the uterus (3), tuberculosis of the testicle (2), gonorrhœa (2), neuritis (3), neurasthenia (14), paraplegia (3), chlorosis (5), malaria (2), angina pectoris (1), Addison's disease (2), Banti's disease (1), cholangitis (17), osteo-arthritis (6), fractured bones (5), alcoholism (2), stricture of the common bile-duct (1), stricture of the cystic duct (1), hydatid of the liver (4), gumma of the liver (1), lead poisoning (1), cystinuria (1), measles (1), scarlet fever (3), rheumatism (1), gout (2). Of the six cases that gave a positive reaction, 3 were suffering from diffuse peritonitis, 1 from pneumonia, 1 was a case of abscess of the liver, and 1 had pyæmia. It is possible that the pancreas was also affected in these cases, for in diffuse peritonitis and pyæmia involvement of the gland is not improbable, while in pneumonia histological changes have been

observed by Chauffard and Ravaut¹ and Trevor,² but the destruction of an excess of nucleo-proteid containing material might also conceivably account for the positive urinary reaction.

(VIII) NORMAL.

In the last group are specimens of urine from 50 presumably healthy individuals, none of which gave a positive pancreatic reaction.

On bringing forward the results of my observations on the urine in diseases of the pancreas in 1904, I stated that "a much more extended experience, dealing with a considerably larger number of cases than it has been possible so far to obtain, will be necessary before the exact value of the test can be determined," and I think that I can now safely say that this has been done. My results during the five years since this was written, which I have collected in this paper, confirm the claim I then made that the reaction is "clinically useful," especially in its improved form. I have repeatedly, and from the first, stated that I do not believe that the "pancreatic reaction" is pathognomonic, or that taken alone it will enable a correct opinion to be formed in every instance; but I do think that, when the results of an examination of the urine are considered in conjunction with the clinical symptoms and an analysis of the fæces, a trustworthy diagnosis is capable of being arrived at in nearly every case of pancreatic disease. I lay considerable stress on the analysis of the fæces, which I shall consider in a separate paper, for from the information so obtained one can infer the extent and often the nature of the pancreatic mischief, and also obtain confirmatory indications as to the cause.

With regard to the nature of the pancreatic reaction itself, my clinical experience and the results of the experiments I have carried out point to its being due to the destruction of substances with a glyco-nucleo-proteid content which yields a pentose on hydrolysis, and, since the percentage of pentose in the dry weight of the pancreas is nearly five times as great as in any other organ of the body, and it is more loosely combined and more readily set free than the corresponding sugar in other tissues, the reaction is obtained with very much greater frequency and more constantly in lesions involving active degenerative changes in

¹ *Arch. de Méd. expér. et d'Anat. path.*, Par., 1901, xiii, p. 175.

² *Practitioner*, Lond., 1904, lxxii, p. 574.

that organ than in others. A single positive reaction cannot be accepted as a conclusive indication of a pancreatic affection, but is strong corroborative evidence of clinical signs pointing in that direction. The affections that are likely to give rise to a positive reaction are usually so readily differentiated from pancreatic disease clinically that there is little or no chance of confusion. Cirrhosis of the pancreas will not give rise to a reaction unless there is also some active inflammation going on at the same time, and that is probably why a negative result is sometimes obtained when the gland is found to be enlarged and hard at operation. So, too, in cancer of the pancreas there is usually no reaction, for no destruction of gland substance takes place, to set free the glyco-nucleo-proteid content of the cells, unless the pressure of the growth or obstruction of the duct sets up a secondary inflammation.

The literature relating to the pancreatic reaction is now somewhat extensive, and it would be impossible to adequately review it at the end of a paper such as this. I may be permitted, however, a few extracts to show the experience and opinions of some independent observers. Kehr recently published the results of his observations on 40 cases before and after operation. He obtained a positive reaction in 82 per cent. of cases recognized clinically as "chronic pancreatitis," and advises that no operation for gall-stones should be undertaken during the latent phase unless the pancreatic reaction is positive, and persists after four weeks' medical treatment. In another series of 58 cases of gall-stones, with secondary injury of the pancreas, the same author operated on 22, and was able to confirm the results of the urinary examination in all but 2, in one of which there was cancer of the common bile-duct. Decker examined 150 urines, and states that a positive result was never obtained "except where the pancreas was under suspicion, or the biliary tract was the seat of the infection." The diagnosis of disease of the pancreas was confirmed in every case where direct examination of the gland was made. Goodman investigated the urines of 62 cases, 10 of which gave a positive reaction. He states that "in no case other than those presenting clinical evidence of disease of the pancreas was a positive reaction obtained. I firmly believe the test to be a very useful one, and to mark a decided advance in the diagnosis of pancreatic disease. It is not pathognomonic, but, taken in conjunction with the clinical history and examination, and careful study of the fæces, a positive reaction is strongly suggestive of inflammation of the pancreas." In another paper the same author, who has also carried out a number of animal experiments, says: "The reaction is almost constantly associated with lesions

of the pancreas. The clinical value of the test seems assured." Kinnicutt obtained a positive reaction with 13 out of 51 cases he examined, and concludes that, "although the test is not pathognomonic, it is strongly suggestive of inflammatory destructive lesions of the pancreas, and is of much assistance in diagnosis in association with other clinical evidence of disease of the organ." Krienitz, who examined 250 cases and operated on 28, found the results of the reaction correspond with the clinical condition in 80 per cent., and adds that it is "a useful indication for diagnosis, prognosis, and treatment." He also carried out a series of comparative observations with Sahli's glutoid test, Schlecht's trypsin test, and the "pancreatic reaction" in the urine, and concluded that the "pancreatic reaction" is especially reliable, as the findings were confirmed by the ultimate course of the cases. Barker, in the course of a paper on cases of pancreatic disease under his observation, concludes: "In my experience, the test is one of the most valuable diagnostic measures ever promulgated." In a discussion on chronic pancreatitis held by the Ohio State Medical Association last year, C. N. Smith stated that "the pancreatic reaction was a valuable and thoroughly reliable indication of the presence of pancreatic inflammation." Levisohn, in the course of his remarks, said "the reaction is of value, and the profession should have recourse to it more frequently"; Schröder concluded that "it is valuable, but not pathognomonic"; and Brown, who had had experience of the test in 39 cases, in 15 of which he had been able to confirm the result, remarked that "the test is one of the most valuable diagnostic aids recently introduced into medicine."

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The Diagnostic Value of an Analysis of the Fæces in Diseases of the Pancreas.

By P. J. CAMMIDGE, M.D.

THE assistance in diagnosis that may be gained by an analysis of the urine in cases of suspected pancreatic disease has been acknowledged for some time by a number of independent observers, but the importance of an examination of the fæces in such cases is not generally recognized. This is partly due to a natural disinclination to undertake such examinations, but is probably also contributed to by the somewhat conflicting statements of different authors who have made analyses. Until the appearance of my paper on "Observations on the Fæces in Biliary Obstruction and Pancreatic Disease,"¹ in which I considered 53 consecutive cases, most of the recorded analyses had only dealt with one or two cases; and it was difficult to draw conclusions from them as the conditions were not identical. Some authors have maintained that an excess of total fat in the fæces is characteristic of disease of the pancreas, while others have failed to find this and considered that faulty cleavage of fats is the important point. Others, again, have stated that an excess of saponified fat is generally met with in pancreatic disease. In this paper, which is based on 579 cases, I shall endeavour to explain the causes of these differences, and to prove that, when taken in conjunction with the clinical symptoms and the results of an analysis of the urine, and particularly the "pancreatic" reaction, a quantitative and qualitative analysis of the fæces may help in the diagnosis of affections of the pancreas, and also indicate their nature and cause.

The points to which I wish particularly to draw attention are: The appearance, the reaction, the proportion of unabsorbed fat, the relation between the unsaponified and saponified fats, the percentage of inorganic ash, the reaction for hydrobilirubin, the presence of occult blood, the so-called pancreatic insufficiency tests, and the bacteriological examination.

APPEARANCE.

The frequent, bulky, soft, white motions of advanced pancreatic disease are very typical, and, to an experienced eye, present characters

¹ *Brit. Med. Journ.*, 1905, ii, p. 1102.

differing from those seen in biliary obstruction, tabes mesenterica, and other disorders with which they might be confused. They are usually whiter, more glistening, and have a peculiar odour resembling that of rancid bacon. Such typical stools are, however, rare; first, because there is very frequently an associated obstruction of the bile flow which alters their characters; and secondly, disease of the pancreas sufficiently advanced to give rise to such typical stools is comparatively uncommon. In the majority of cases of pancreatic disease, apart from those associated with biliary obstruction, the fæces show no characteristic change to the naked eye, even when chemical analysis reveals considerable alteration in their composition.

REACTION.

The reaction of the normal fæces is amphoteric, faintly alkaline or faintly acid. A strongly alkaline or acid reaction is pathological. As a rule the stools in pancreatic disease are acid, the intensity of the reaction depending upon the degree to which the digestive functions of the gland are interfered with. I have met with two cases with markedly acid stools, in which the irritation of the lower bowel was so great that it had to be washed out several times a day to allay the discomfort. In some cases the reaction is either amphoteric or alkaline, occasionally strongly alkaline, but in these the pancreatic disease is associated with biliary obstruction, in which the stools are usually alkaline, or is secondary to an intestinal catarrh, which generally gives rise to fæces with a markedly alkaline reaction.

MICROSCOPICAL EXAMINATION.

Microscopical examination of the bulky white stools of advanced pancreatic disease shows numerous fat globules, fatty acid crystals, and undigested muscle fibres. Muscle fibres, provided that the patient is not an excessive meat eater and the digestive functions of the stomach are being normally carried out, are particularly characteristic, and are best and most frequently seen in cases of malignant disease of the pancreas. Fat in the form of undigested globules is also fairly characteristic, but it must be remembered that fatty stools are also met with in biliary obstruction, apart from disease of the pancreas, in diseases of the intestinal tract where absorption is interfered with, such as sprue, tuberculosis or malignant disease of the intestine, where the retro-peritoneal lymph glands are involved and there is blocking of the lymph

flow, and in persons on a milk diet or who are taking much cod-liver or olive oil. The earlier stages of diseases of the pancreas produce little or no change in the microscopical characters of the stools.

CHEMICAL ANALYSIS, FATS.

Since the preparation of fats for absorption by the intestine is peculiarly a function of the pancreatic juice it is to an investigation of the undigested fat in the fæces that attention has been chiefly directed in searching for signs of disease of the pancreas. The method I employ is that which I first described in 1905.¹ It is much quicker and easier to apply than those in general use, and gives satisfactory results for diagnostic purposes. In this method the total fat, calculated as a percentage of the dry weight of the fæces, is first estimated by extraction with ether, after boiling with dilute hydrochloric acid. The unsaponified or undigested fat, including the unaltered fat and free fatty acid, is then determined in a second sample by extracting a watery suspension of a known weight of the dry fæces with ether. The difference between the two gives the proportion of saponified fats, or soaps, which have undergone digestion.

TOTAL FAT.

The percentage of total fat in the dry weight of the fæces in cases of disease of the pancreas has been found to vary very much by different observers. This has been partly due to their investigating different forms and stages of pancreatic disease, and to their not making allowance for the causes of the disease and the modifications that these may bring about in fat digestion and absorption. I have endeavoured to overcome this difficulty by arranging my cases in groups according to the cause as far as possible (*see* Table I). A glance at the table will show that very wide variations in the percentage of total fat have been met with, not only in different forms of pancreatic disease but also in patients suffering from the same disorders. The average percentage has, however, been abnormally high in nearly every class. The highest percentage has been encountered in malignant disease of the pancreas, 93·3 per cent. of the dry weight of the stool examined consisting of unabsorbed fat in 1 case. Here there was complete blocking of the

¹ *Brit. Med. Journ.*, 1905, ii, p. 1102; and "The Pancreas: Its Surgery and Pathology," 1908, p. 211.

TABLE I.—

[illegible]

PANCREATIC DISEASE.

UNSAAPONIFIED		ASH				HYDROBILIRUBIN			Blood	REACTION		
Greater than saponified fat	Less than saponified fat	Average	Excess	Normal	Sub-normal	Present	Traces	Nil		Acid	Neutral	Alkaline
29	21	12.6	9	28	14	43	8	0	2	29	2	20
14	11	14.5	11	12	2	25	0	0	0	14	2	9
6	5	18.5	7	2	0	11	0	0	0	3	1	7
1	1	10.9	0	2	0	0	2	0	1	0	2	0
1	—	12.4	—	—	—	—	1	—	0	1	—	—
25	58	16.3	49	29	5	83	0	0	0	8	18	57
14	10	12.1	8	10	6	24	0	0	0	14	6	4
7	8	15.4	6	6	1	15	0	0	3	4	2	9
1	2	15.9	2	1	0	3	0	0	1	0	1	2
4	4	13.3	2	5	1	8	0	0	0	6	1	1
0	2	26.2	2	0	0	2	0	0	0	0	0	2
1	—	12.0	—	1	—	1	—	—	0	—	1	—
8	1	11.8	1	5	3	9	0	0	0	5	4	0
8	0	12.4	2	3	2	8	0	0	0	4	3	1
4	4	17.9	4	3	1	8	0	0	1	2	4	2
3	1	16.4	3	1	0	4	0	0	1	1	0	3
4	0	7.4	0	0	4	2	2	0	2	1	1	2
3	5	19.0	5	3	0	8	0	0	6	1	2	5
2	4	16.5	4	2	0	6	0	0	5	0	1	5
3	1	14.3	0	3	1	4	0	0	0	0	1	3
23	24	15.9	24	20	0	47	0	0	4	13	6	28
1	—	12.6	—	1	—	1	—	—	0	1	—	—
1	—	12.6	—	1	—	1	—	—	0	1	—	—
22	16	9.0	1	11	26	3	13	22	25	30	0	8

common bile-duct, so that no trace of bile pigment found its way into the intestine, and the very high proportion of fat was, no doubt, in part due to this. The comparatively low percentage of 22·3, found in 1 case of cancer of the pancreas, is to be explained by the growth being situated in the tail of the gland, so that the greater part of the organ was able to carry out its functions in a normal manner. Such cases are rare, and the fact that an average percentage of 71·3 of total fat was found in my 38 cases shows that a marked excess of unabsorbed fat is the rule.

Chronic pancreatitis appears in severe cases to interfere with fat digestion and absorption almost as much as malignant disease of the gland, for 87·2 per cent. was found in 1 case where there was obstruction of the common bile-duct due to impacted gall-stones, and 72·2 per cent. in another where there was marked cirrhosis of the pancreas but no obstruction of the bile flow. Such high readings are not common, the average in 51 cases of chronic pancreatitis associated with gall-stones in the common duct and jaundice being 56·6 per cent., and in 8 cases of cirrhosis of the pancreas 36·2 per cent. Muller has attributed the steatorrhœa in pancreatic cases with jaundice entirely to absence of bile.¹ Although a very considerable increase of fat in the stools may result from simple biliary obstruction the increase is usually greater when there is an associated affection of the pancreas; thus, in 16 cases of common duct obstruction without evidence of pancreatitis I found that the average amount of total fat was 54·8 per cent., the highest reading being 81·3 per cent., and the lowest 25·7 per cent., whereas in the 51 with pancreatitis that I have already mentioned the average was 56·6 per cent., the highest being 87·2 per cent., and the lowest 15·8 per cent. The results obtained in cases of floating stones in the common duct are interesting in this connexion, for here there is no interference with the bile flow. In 25 such cases, where there was pancreatitis, the average reading of total fat was 36·5 per cent., with a maximum of 71·3 per cent. and a minimum of 16·0 per cent., and 9, where no evidence of secondary pancreatic mischief was found, showed an average of 33·2 per cent., with a maximum of 62·3 per cent. and a minimum of 21·2 per cent.

On looking down the table it will be noticed that, although the average of total fat is nearly always abnormally high in every group of case in which disease of the pancreas was present, and the highest readings obtained were very markedly in excess of the normal limit of

¹ *Zeitschr. f. klin. Med.*, Berl., 1887, xii, p. 45.

about 25 per cent., the lowest were not infrequently much under the normal limit of about 10 per cent. This is at first sight contrary to what might be expected, but the explanation is that these were cases in an early stage where as yet the secretion of the pancreatic ferments had not been interfered with, but was in all probability rather increased, for just as in the early stages of parotitis there is an increased flow of saliva, so in catarrhal pancreatitis there is probably also an increased flow of pancreatic juice, so that fat digestion is rather increased than diminished. In support of this supposition it may be pointed out that the lowest readings were obtained in cases of intestinal catarrh, duodenal or gastric ulcer, and catarrhal jaundice, in which it is probable that the pancreatitis was due to catarrhal inflammation spreading from the duodenum along the pancreatic ducts. In these cases, however, the abnormal activity of fat-splitting bacteria in the intestine has also to be taken into account and may afford an additional explanation.

RELATION OF UNSAPONIFIED TO SAPONIFIED FATS.

The presence of an excess of unabsorbed fat in the fæces is suggestive of pancreatic disease, but a normal and even a sub-normal proportion does not exclude it, especially in the early stages of a catarrhal inflammation. Steatorrhœa is also met with in other diseases, such as simple biliary obstruction, tuberculosis of the intestine, &c., and may arise from defective gastric digestion or an excess of fat in the food, so that alone it is not of great diagnostic value. The relation existing between the percentages of unsaponified and saponified fats is much more important, and even when an abnormally low percentage of total fat is present in the stool, a disturbance of this relation may help to confirm a diagnosis of disease of the pancreas. In normal fæces the unsaponified and saponified fats are present in approximately equal amounts, and any marked change in this relation is indicative of disturbance of digestion. The normal percentage of each varies from about 10 to 15 per cent. The effect of interference with the functions of the pancreas is, as might be supposed from a consideration of its work in digestion, to increase the proportion of unsaponified fat, while obstruction of the bile flow and intestinal catarrhs tend to raise the percentage of saponified fat. The effect of disease of the pancreas in this direction is well seen in the 8 cases of cirrhosis of the gland, for in all of these the unsaponified were in excess of the saponified fats, the former averaging 22·6 per cent. and the latter 13·6 per cent., or again in the

case of cyst of the pancreas where, although the total fat was only 20·5 per cent., nearly three-quarters of this (15·0 per cent.) was in the unsaponified and only about a quarter (5·5 per cent.) in the saponified form. A marked difference between the proportions of unsaponified and saponified fats is seen in the case of pancreatic infantilism, the former working out at 48·5 per cent. of the dry weight, and the latter at 8·9 per cent. On the other hand, in a case of simple stricture of the common bile-duct (Table II) I found 52·1 per cent. of the dry weight of the fæces consisted of saponified and only 27·8 per cent. of unsaponified fat, and in 16 cases of jaundice due to obstruction of the common duct by gall-stones, the proportion of saponified exceeded that of unsaponified fat in 12, and was only equal to or less than it in 4, the jaundice in these 4 being not very marked and the difference slight. The average percentage of unsaponified fat in these 16 cases was 21·7 per cent. and of saponified fat 33·1 per cent.

The effect produced by co-existent pancreatic disease and biliary obstruction depends upon the relative extent and standing of the two conditions. In cancer of the pancreas, where as a rule there is complete blocking of the common bile-duct and serious interference with the functions of the pancreas, since the growth is most commonly situated in the head of the gland, the proportions of unsaponified and saponified fat are frequently nearly equal, although in 22 out of 38 cases I found an excess of the former, the percentage varying from 69·0 to 7·0 per cent. with an average of 41·0 per cent. for the unsaponified, and from 63·8 to 3·6 per cent. with an average of 30·3 per cent. for the saponified fat. Chronic pancreatitis due to the presence of gall-stones in the common bile-duct and associated with obstructive jaundice gave an excess of unsaponified fat in 29 of my cases, and an excess of saponified fat in 21. Here again very wide variations in the percentages of each were met with, depending apparently upon the extent to which the bile flow was interfered with and the amount of damage of the pancreas. An average of 30·2 per cent. was found in these 51 cases for the unsaponified fats and 26·4 per cent. for the saponified fats, the former ranging from 59·8 to 5·5 per cent., and the latter from 55·8 to 2·3 per cent.

I have examined the fæces from 7 cases of growth of the common bile-duct. In 5 of these there was no evidence of involvement of the pancreas, and the saponified fat (average 44·0 per cent.) was considerably in excess of the unsaponified fat (average 30·7 per cent.). Of the 2 in which the pancreas was involved in the growth, and the urine

gave a well-marked pancreatic reaction, one showed an excess of saponified and the other of unsaponified fat, the readings for the one being 33.2 and 39.5 per cent., and for the other 25.9 and 0.3 per cent. for the unsaponified and saponified fats respectively, but the last-named case was on an entirely milk diet, so that the results are not strictly comparable with the others, as the experiments of Abelman¹ have shown that in depancreatized animals from 30 to 53 per cent. of a natural emulsion such as milk is absorbed, and that when a portion of the gland has been left as much as 80 per cent. may be digested.

Another factor which plays an important part in determining the relation between the unsaponified and saponified fats in the fæces is the distribution and activity of the intestinal flora. Owing to the defensive action of the stomach juices and the rapid passage of the chyme through the upper intestine in a healthy person comparatively few bacteria are met with until within a foot or two of the colon. In this region, and in the large intestine, a certain amount of fat saponification normally goes on from the action of bacteria of the colon group. In acute and chronic pathological conditions of the gastro-intestinal tract the altered state of the secretions allows an ascent of micro-organisms above the level of their normal habitat,² and, owing to their increased number and probably also to alterations in their chemical activities, the saponification of fats is more energetically carried out, so that in cases of gastro-intestinal catarrh it is the rule to find a more or less marked excess of saponified over unsaponified fat in the fæces. Thus in 18 cases diagnosed as "intestinal catarrh or enteritis" without pancreatitis and a negative pancreatic reaction, such an excess was found in all but one; also in all of the 6 cases of duodenal ulcer, the 13 of gastric ulcer, and the 3 of appendicitis without evidence of pancreatic disease, a similar relation was found to exist. Even when pancreatitis is present and there are other indications that the digestive functions of the gland are interfered with, the excess of unsaponified fat that might be expected is often replaced by an abnormally high percentage of soaps owing to the fat-splitting action of the intestinal bacteria. Out of the 83 cases of pancreatitis associated with intestinal catarrh that I have examined the saponified fats were found to be in excess in 58, and in only 25 were the unsaponified and saponified fats equal or the former in excess. It is obvious that when there is interference with the functions of the pancreas from cirrhosis consequent on

¹ "Dissertation," 1890.

² Herter, "The Common Bacterial Infections of the Digestive Tract," New York, 1907, p. 265.

PANCREATIC DISEASE.

UNSAAPONIFIED		ASH				HYDROBILIRUBIN			Blood	REACTION		
Greater than saponified fat	Less than saponified fat	Average	Excess	Normal	Sub-normal	Present	Traces	Nil		Acid	Neutral	Alkaline
4	12	12.2	3	10	3	14	2	0	0	4	1	11
4	5	18.2	7	2	0	9	0	0	0	0	3	6
11	12	15.5	7	8	3	23	0	0	0	1	6	16
0	5	10.6	0	4	1	2	2	1	3	3	1	1
—	1	13.6	—	1	—	—	1	—	0	—	1	—
1	17	20.3	18	0	0	18	0	0	0	0	2	16
0	2	32.6	2	0	0	2	0	0	0	0	0	2
0	6	19.0	5	1	0	6	0	0	2	0	0	6
0	13	16.1	8	4	1	13	0	0	4	0	0	13
2	3	13.3	1	4	0	5	0	0	0	5	0	0
1	—	13.4	—	1	—	1	—	—	0	—	1	—
0	3	16.8	3	0	0	3	0	0	0	0	0	3
0	30	25.3	30	0	0	30	0	0	5	0	0	30
0	3	20.4	3	0	0	3	0	0	0	—	1	2
4	1	14.8	3	2	0	5	0	0	0	0	2	3
0	4	24.1	3	1	0	4	0	0	0	0	0	4
0	2	13.7	0	2	0	2	0	0	0	0	0	2
5	10	14.5	8	5	2	15	0	0	12	0	10	5
1	18	19.0	18	1	0	19	0	0	17	0	5	14
4	6	13.1	0	8	2	10	0	0	0	0	4	6
12	13	12.5	0	25	0	25	0	0	0	2	20	3

inflammatory changes, and also an abnormal activity of the fat-splitting bacteria of the intestine, the relation between the saponified and unsaponified fats will vary with the relative intensity of the two, and no hard-and-fast rule can be laid down, each case must be judged on its merits, and the indications to be obtained by other methods of examination. Of these the presence of indicanuria, an excess of inorganic ash in the fæces and the results of the pancreatic insufficiency test are the most important. The varying relations found in my cases of duodenal and gastric ulcer are examples of this, and the same explanation probably holds good for the cases of sprue and pernicious anæmia that I have examined. Stones in the common bile-duct and gall-bladder have frequently not been found to be associated with a disturbance in the relation between the saponified and unsaponified fats in the direction that might theoretically be expected, especially when there has been no obstruction of the bile flow, and I am inclined to think that this may be due to the abnormal activity of fat-splitting bacteria, for in such cases there is generally evidence of cholangitis and this, as well as the gall-stone formation, is probably consequent upon invasion of the biliary tract by intestinal organisms which have ascended beyond their normal limit to the level of the common bile-duct.

When speaking of the total fat in the fæces I mentioned that in the early stages of catarrhal pancreatitis there is probably an increased flow of pancreatic juice which may bring about an abnormally low reading. In such cases it is the rule to find that the saponified are in excess of the unsaponified fats, often very markedly so, even when there is no evidence of abnormal bacterial activity in the intestine. This is probably due to the excess of pancreatic juice causing a higher proportion than normal of the fats to undergo saponification and, although much of this is absorbed, leading to a low total fat reading, a higher proportion of soaps than usual appears in the fæces.

INORGANIC ASH.

The excretion of calcium and magnesium salts appears to take place mainly by way of the intestinal mucous membrane, and particularly through the large intestine, the amount passed in the urine being comparatively small. Normally the proportion of inorganic ash in the fæces lies between 10 and 15 per cent., but when from any cause there is a catarrh of the intestinal walls this percentage is increased, and I have met with readings as high as 45 and 46 per cent. These have usually

been cases where there has been well-marked chronic colitis. The occasional association of intestinal sand with muco-membranous colitis, and more rarely with diarrhoea, was pointed out by Dieulafoy, but I believe I was the first to show that an increase in the proportion of inorganic ash occurs in all cases of colitis.¹ Such an increase was found in each of the 30 cases of colitis that I have examined, the average for the 30 being 25·3 per cent. In appendicitis, intestinal catarrh, and growth of the intestine, particularly of the colon, a similar, although less marked, increase has usually been met with, probably because in these, too, there is some colitis. For, although clinically the disease may appear to be more or less localized in some particular part of the intestine, it is not unlikely that other regions are also involved to a less extent, and that this is so is suggested by the occasional association of pancreatitis, as indicated by the urinary reaction, with appendicitis and of chronic colitis with enteritis or duodenal ulcer. An excess of inorganic ash in the fæces is therefore indicative of an intestinal catarrh, and more particularly of a catarrh of the colon. In such cases the stools are usually strongly alkaline in reaction.

BILE PIGMENT.

Bile is the main ingredient that takes any considerable part in furnishing the colour of the stools. Its chief pigment as excreted is bilirubin. A part of this is promptly oxidized, either in the bile passages or soon after it reaches the intestine, into biliverdin and allied bodies, and, under the influence of bacteria and enzymes in the intestine, these are reduced to hydro-bilirubin (stercobilin), which constitutes the normal yellowish-brown pigment of the fæces. Bile pigment, as such, never appears in the normal fæces, and in health is not met with below the cæcum. Interference with the bile flow is, therefore, an important factor in producing alterations in the colour of the fæces, the well-known "clay-coloured" stools of biliary obstruction being the result. Since many cases of pancreatic disease in which white stools exist are associated with more or less complete blocking of the common bile-duct by gall-stones, or growth in the head of the pancreas, the lack of bile pigment is without doubt a frequent contributory cause of their production. I have, however, met with cases of pancreatitis with white stools where there was no jaundice or biliary obstruction, and in which a chemical

¹ *Med. Chir. Trans.*, Lond., 1907, xc, p. 616.

examination of the stools showed a well-marked reaction for stercobilin, also others in which it was demonstrated that a return of the biliary secretions to the intestine by means of a cholecystenterostomy was not sufficient to bring about a return of the normal colour when the pancreatic juice was still absent. Both in these and the clay-coloured fæces of simple biliary obstruction the large excess of unabsorbed fat is an additional and important cause of their abnormal physical characters.¹ The whiter, more glistening appearance of the fæces in typical cases of pancreatic disease is, I believe, to be attributed to the existence of the fat largely in the form of free fatty acid crystals, which act upon light in the same way as snow and other finely crystalline substances that appear white in mass. Another factor which must not be overlooked, and which plays an important part in the production of the pseudo-acholic stools met with in other conditions where there is no icterus and possibly also no interference with fat digestion and absorption, is the action of bacteria on the fæcal pigments. A certain amount of the hydrobilirubin of the fæces frequently, and perhaps always, undergoes further reduction to a colourless body, called by von Nencki leucourobilin, through the action of bacteria, and it is probable that under pathological conditions this reduction may be so marked that little or no coloured pigment remains.² I have shown by a series of experiments that the bacteria present in the white stools of pancreatic disease have the power of decolorizing normal brown fæces when grown under anaerobic conditions,³ and I believe that it is to the abnormal activity of such bacteria that the absence of colour is due to a considerable extent in such cases.

It is obvious from these considerations that mere inspection of the stools is not a reliable guide as to the amount of bile pigment that is reaching the intestine, and for purposes of diagnosis this is most important to determine. Hydrobilirubin gives the same chemical reactions as the urinary pigment urobilin, with which it is closely allied, and it is only by applying these chemical tests that the presence of complete biliary obstruction can be determined with certainty. The test I usually employ is the production of a green fluorescence when an acid-alcohol extract of the fæces is neutralized and mixed with an alcoholic solution of zinc acetate.⁴

¹ See "The Pancreas: Its Surgery and Pathology," 1908, p. 226.

² Herter, "The Common Bacterial Infections of the Digestive Tract," 1907, p. 294.

³ "The Pancreas: Its Surgery and Pathology," 1908, p. 227.

⁴ *Ibid.*, 1908, p. 213.

A more or less marked reaction for hydrobilirubin was obtained in all my cases, except 22 of malignant disease of the pancreas, and 1 of cancer of the common bile-duct, even when the fæces was quite white to the naked eye. In those where there was biliary obstruction from gall-stones, the intensity of the reaction varied with the degree of jaundice, but traces at least were found in every one, and in all but 10 a fairly well marked reaction was obtained, showing that rarely, if ever, does impaction of gall-stones in the common bile-duct produce absolute blocking. On the other hand, out of 38 cases of cancer of the pancreas complete obstruction of the duct was met with in 22, traces of hydrobilirubin were found in 13, and in 3 only was a well-marked reaction obtained, and in these 3 the growth was situated in the tail or body of the gland. As a rule, malignant disease of the common bile-duct and gall-bladder does not produce the complete blocking of the bile flow usually associated with cancer of the head of the pancreas, probably because the growth in these cases is of a soft character, and allows a certain amount of bile to percolate through it. Out of 7 cases of growth of the common duct a well-marked reaction for hydrobilirubin was obtained in 2, traces in 4, and no reaction in 1. Six cases of growth of the gall-bladder showed a well-marked reaction in 4, and traces in 2.

BLOOD.

In the last few years considerable attention has been devoted to the examination of the fæces for occult blood, that is to say, for traces of blood too small to be recognized by other than chemical means, and for this purpose the guaiac, aloin, and benzidine tests have been chiefly employed. The guaiac and aloin tests are about equal in sensitiveness, both responding to dilutions of 1 to 25,000, and are useful as controls for the simpler and more sensitive benzidine test, which reacts with 1 part to 200,000 of blood. In carrying out these tests it is important that the correct procedure should be adopted, and that disturbing factors such as a diet rich in meat, iron salts, oxidizing ferments, whether animal or vegetable, potassium iodide, &c., should be excluded. If the benzidine test is negative, it may be concluded that no blood is present, and it is not necessary to proceed further, but if it is positive it is advisable that the result should be controlled by the aloin or guaiac test. If these are also positive the presence of occult blood is indicated, but if, on the other hand, they are negative, the presence of occult blood should be regarded as

doubtful, and a further examination be made on a future occasion. Hence a positive benzidine test shows nothing unless corroborated by one or both of the other tests, but a negative benzidine test is proof positive of the absence of occult blood.¹

I have examined 579 specimens of fæces with these tests, and obtained evidence of the presence of blood in 89. It has been said that occult blood is found in the fæces constantly in malignant disease of the gastro-intestinal tract, whereas in simple ulceration it is only met with at intervals. My experience would tend to confirm this, for out of 25 cases of malignant disease of the intestine I found it in 22; in 23 of growth of the stomach it was present in 18; but in 21 cases of duodenal ulcer a positive result was only obtained in 5, and in 16 cases of ulcer of the stomach in 5. In several of the latter a series of five or six examinations on succeeding days was made before a trace of blood was found. Cancer of the pancreas is very frequently associated with the presence of occult blood in the stools. In 25 of my 38 cases a positive result was obtained. The hæmorrhagic tendency that is known to be present in chronic pancreatitis is sometimes shown by the presence of blood in the fæces, and in advanced cases serious hæmorrhage may take place from the intestinal and other mucous membranes. In two of my cases such hæmorrhage was the cause of death. This hæmorrhagic tendency is the probable explanation of the positive reaction for blood given by the stools of the two cases of chronic pancreatitis associated with jaundice, and the presence of gall-stones in the common duct, and the 4 cases of pancreatitis in which there was no biliary obstruction. I was able to detect blood in the fæces of 4 out of the 7 cases of malignant disease of the gall-bladder, and in 2 out of the 6 cases of growth of the common duct that I have examined. The blood found in the cases of arteriosclerosis and heart disease was due, in one instance, to small ulcers at the cardiac end of the œsophagus, and in the other was probably the result of venous congestion and back pressure.

PANCREATIC INSUFFICIENCY TESTS.

Early in the year 1908 Schlecht² described a method of estimating the functional activity of the pancreas, based upon the observation

¹ Goodman, *Amer. Journ. of Med. Sci.*, Philad., 1907, cxxxiv, p. 506.

² *Münch. med. Wochenschr.*, 1908, lv, p. 725.

of Müller,¹ that it was possible to detect the presence of trypsin in the fæces by its action on a serum plate. I have employed this test in a number of cases and have found that on the whole it is satisfactory, but the necessity for using a serum plate is a drawback, and it is not always easy to determine the amount of change that has taken place. I have, therefore, been using lately a modification introduced by Gross,² in which these difficulties do not occur. Gross's test is based upon the fact that pure casein, which is readily soluble in an alkali, is promptly precipitated by acetic acid from such a solution, in contrast to its digestion products. With the solutions and proportion given in the original paper, casein digestion is generally complete, at body temperature, in eight to fifteen hours, but it is sometimes longer, and a normal limit of thirty hours has been set. I have employed this test in about 60 cases of suspected pancreatic disease, and find it a useful additional aid in diagnosis. In 2 cases of cancer of the pancreas the precipitate with acetic acid at the end of thirty hours' incubation was as dense as that given by a control specimen that had not been incubated, and in 2 cases of diabetes very little digestion appeared to have taken place in the same time. In 5 cases of stone in the common duct, with pancreatitis and jaundice, a fairly well marked precipitate was obtained at the end of thirty hours, but it was not as dense as in the cancer and diabetes cases. On the other hand, several cases of catarrhal pancreatitis, with intestinal catarrh, appeared to produce abnormally rapid digestion, for no precipitate could be obtained after six hours' incubation. In 3 cases of diabetes, and in a number of patients suffering from diseases not apparently involving the pancreas, casein digestion was complete within the limit of time regarded as normal.

More recently Heiberg³ has introduced a modification of Gross's test, in which a series of progressive dilutions of the fæcal extract are incubated for a fixed time of ten hours, with the same quantity of casein solution. This method has the merit of shortening the time of the examination, and also enables the various samples to be examined together and compared. I have made use of this test, along with Gross's method, in the last 30 specimens that I have investigated, and I find that the results compare very well. For the

¹ *Archiv f. klin. Med.*, Leipz., 1908, xcii, p. 199.

² *Deutsch. med. Wochenschr.*, 1909, xxxv, p. 706.

³ *Wiener. klin. Wochenschr.*, 1909, xxii, p. 52.

reasons I have mentioned I think that Heiberg's method is to be preferred.

It must be remembered that these tests only indicate very gross changes in the digestive functions of the pancreas, and so are only of limited value when taken alone, but when considered in conjunction with other methods of examining the fæces and urine they certainly assist one in arriving at a more reliable opinion as to the condition of the gland.

BACTERIOLOGICAL.

In spite of the work of a large number of observers the bacteriological examination of the fæces, as an aid to diagnosis, is as yet in its infancy. In certain diseases it can no doubt be carried out with advantage, but the methods are too complicated and lengthy for routine work. The labours of Herter, Kendall, and others give promise that in the future they may be sufficiently simplified for a bacteriological examination to form part of the investigation of the fæces in all cases of gastro-intestinal disease. At present the most helpful and generally applicable method is the examination of cover-glass preparations, treated with Gram's stain. By the use of Gram's method one may obtain information as to the presence or absence of certain types of bacteria in the digestive tract, and, after one has had some experience, form an opinion as to whether spore-bearing organisms and vegetative anaerobic types are present in excessive numbers. Such microscopical appearances cannot be regarded as positive evidence of the identity of the dominant bacteria present in the intestine, but they may aid in forming conclusions that are valuable on account of their high degree of probability, and serve to confirm a diagnosis of intestinal disease arrived at by other methods. It is, however, essential that examinations of this nature should be based upon a thorough acquaintance with the appearance of the normal fæces, and that the variations brought about by differences in diet should be remembered.

I take this opportunity of thanking those who have kindly sent me the cases on which this paper is based, and for their replies to my inquiries concerning their course and the ultimate diagnosis.

DISCUSSION.

The PRESIDENT (Dr. Mitchell Bruce) said he was sure that all present would join in thanking Dr. Cammidge for his two papers, and in an expression of admiration of the work which they contained. One could not help being struck, not only by the amount of work actually done by him, but by the variety of circumstances under which that work had been accomplished. The number of individual facts and the wealth of material presented made it somewhat difficult to discuss the work, and no one present could draw upon anything like the same number of cases. Still, the Section would be glad to hear from those who had used the method.

Dr. ROLLESTON wished to re-echo the President's admiration of the amount of work which Dr. Cammidge had devoted to the tests during the last ten years. He (the speaker) could not say anything with regard to the chemical questions involved in these tests, but he could testify to the value of the Cammidge tests in establishing the diagnosis in cases in which this might not be possible by ordinary clinical methods. Though it might be easy to distinguish between ordinary characteristic cases of stone in the common duct and those in which the jaundice was due to malignant disease, in which operation was not desirable, yet there were a number of cases in which the clinical manifestations so departed from the normal, or were so confused, that it was impossible to decide with certainty whether a case of jaundice was due to gall-stones, to a growth in the head of the pancreas, to carcinoma of the common bile-duct, or to a growth in the portal fissure. He had seen cases in which Cammidge's tests had shown that a jaundice which had come on gradually, without pain, and was deep, and therefore resembled the jaundice caused by malignant disease, was really due to a stone in the common duct. In such cases a positive reaction of Cammidge's tests, pointing to stone in the common duct, had justified an exploratory operation, which had proved eminently successful. Another point of interest was the frequency with which a pancreatic reaction occurred in cases in which it was not suspected clinically—cases, for example, in which there was no jaundice, and in which the symptoms did not suggest that there was anything the matter with the biliary apparatus. Some of the cases he had seen had been in doctors of middle age who had had gastro-intestinal disturbance, shown by colicky pain, precipitate and explosive diarrhœa, and often by extremely offensive motions. In these cases the infection had probably spread from the intestine to the pancreas. It did not follow that because there was a positive pancreatic reaction the operation for the relief of pancreatitis—namely, draining the gall-bladder—should be performed in these cases. He had seen cases in which Cammidge's tests pointed to the presence of pancreatitis, in which operation was suggested though not carried out, and in which the patient eventually recovered. He did not for a moment suggest that Dr. Cammidge considered that all cases with a positive pancreatic reaction should be operated upon.

Dr. McCULLOCH wished to join in thanking Dr. Cammidge for his paper. One matter apropos of this subject which he had not touched upon had been the mechanisms in connexion with the gall-bladder and pancreas, on which subject there was a surgical address delivered at the seventy-third annual meeting of the British Medical Association, at Leicester, by Mr. C. J. Bond. It was entitled "On Ascending Currents in the Mucous Canals and Gland Ducts, and their Influence on Infection: A Study in Surgical Pathology."¹ Mr. Bond referred to the upper intestinal tract in particular. His experiments were made by means of indigo given in cachets and were convincing. The indigo passed the small intestine without being taken up by any reverse current in those ducts, the common bile-duct, and the pancreatic duct, except when a fistulous condition existed from any cause, or there was a plug of mucus in one or other ducts causing a reverse current in those ducts, such currents carrying with them micro-organisms or vegetable matter, such as cellulose, which he suggested went to form condensation nuclei for the subsequent formation of gall-stones.

Dr. HERTZ desired to inquire why Dr. Cammidge did not order some special diet, such as that of Adolf Schmidt, before making his analysis of the fæces. He would have thought that with so many possible varieties of diet the tests would lose much of their value. With regard to the cases of catarrhal jaundice, he asked whether the cases in which reaction was negative were those in which recovery took place most rapidly. He supposed that when a case of jaundice was so severe that it required operation, as it had done in several of Dr. Cammidge's patients, most people would hardly be inclined to include it as a case of catarrhal jaundice. His own impression would have been that the mild cases of jaundice commonly referred to as catarrhal were more likely due to catarrh of the duodenum than an ascending affection of the pancreas, and that the latter occurred only in more severe cases, including those in which the question of operation might possibly be discussed.

Dr. CAMMIDGE, in reply, expressed his gratification to those who had spoken. The work had occupied him constantly during the last ten years, and had been carried out in the face of many difficulties and much adverse criticism, which, on the whole, was salutary, as it prevented one from falling back into a slothful state of mind. In the paper he had included a list of all the contributions he could find dealing with pancreatic reaction, and the work of most other observers seemed to have largely coincided with his own. Dr. Chalmers Watson had written in the *British Medical Journal* that he regarded the so-called pancreatic reaction as a complicated process, which it required some experience to carry out satisfactorily. There were certainly many details which, if not carefully attended to, interfered with the success of the test as a diagnostic measure. There was nothing more difficult than to write out a description of a chemical process in such a way that no one who attempted to do it could possibly make a mistake. In three recent publications which had given a description of the method of carrying out the pancreatic

¹ *Brit. Med. Journ.*, 1905, ii, p. 232.

reaction there was a mistake which would lead to the vitiation of the result. One gentleman said the urine must be boiled for ten seconds, whereas it should be ten minutes; another said hydrochloric acid of a specific gravity of 1.19 must be used, but it should be 1.16; a third advised the use of lead acetate instead of tribasic lead acetate, which is a very different thing. One doctor told him that at the laboratory where he was working they got a positive reaction in every urine they examined. He (Dr. Cammidge) replied that he got many negatives, and he would like to see how it was done. He did so, and found that each specimen was measured in the same measure glass without washing it out, so that it carried through some of the unhydrolysed urine, and the glycuronic acid was not therefore completely precipitated out by the tribasic acids; consequently a positive reaction was obtained every time. It was advisable that the test should be carried out by someone who had had a proper training in exact chemical methods. With regard to Dr. Rolleston's remark about malignant disease of the pancreas and stone in the common duct, it was to distinguish those conditions that the work was originated. The test had not quite turned out in that respect as had been hoped, and it was only by taking the pancreatic reaction and the results of a quantitative analysis of the fæces together that one could arrive at a correct diagnosis. The only condition which could not be readily differentiated was malignant disease of the ampulla from stone in the common duct. But of the former he had had only three cases in ten years. Two months ago he had a case which was diagnosed by a surgeon as malignant disease in the head of the pancreas in the early stage. The patient was deeply jaundiced, and he (Dr. Cammidge) thought it was pancreatitis due to stone in the common duct. Operation showed there was obstruction to the common duct, but that it was due to a growth in the ampulla, which was producing the same effect as a stone. Dr. Rolleston had said that many doctors suffered from indigestion of sorts. He (the speaker) scarcely passed a week without seeing two or three medical men about troubles of digestion, and the history was nearly always the same—they had a busy practice, they bolted their food, and perhaps often had to rush off in the middle of a meal. They generally started with symptoms of hyperchlorhydria, followed by distension of the intestine, with alternating constipation and diarrhœa, and the symptoms of intestinal indigestion. They had, in fact, a catarrhal condition spreading all the way through the gastro-intestinal tract which involved the pancreas and its ducts, and often also the bile-ducts, as a result of irregularity of meals. The imperfect digestion was primarily gastric and intestinal, but it was intensified and eventually kept up by the pancreatic changes that followed. It was such a regular story that he could generally tell the patient beforehand what his history would be. In old-standing cases there was sometimes glycosuria, and this was the great danger. He did not give any special diet before analysing the fæces, as he thought it a mistake. When making an analysis of the fæces, one wanted to know what the patient's intestine and stomach could do with the food he was in the habit of taking—not what he could do with some abnormal and often distasteful mixture such as the average

test meal. If one gave the patient a mixed diet of ordinary foods, satisfactory conclusions could be arrived at, particularly since it was not usually the total amount of fat which was the important thing, but the way in which the cleavage of fats was carried out. With regard to catarrhal jaundice, most people called a condition by that name when they did not know what else to call it. If a patient had jaundice, and had not pain suggestive of gall-stones, or symptoms suggesting malignant disease, it was called catarrhal jaundice, and those were the cases he included in his paper. The majority of these cases he had not seen personally, and only made an analysis of specimens sent for his opinion. Three or four months afterwards, however, he always wrote asking for a form to be filled up saying what the symptoms were subsequent to his investigations, and what the final diagnosis was. In the cases of which he had personal cognizance the pancreatic reaction was not usually given where the jaundice quickly cleared up. Where the jaundice persisted for some time there was a pancreatic reaction, and where the pancreatic reaction persisted the jaundice persisted too. Those were the cases where it was often advisable to suggest operation to allay the inflammation at the head of the pancreas. He thought that to operate in every case of pancreatitis was unnecessary. There were only a few cases in which operations were required, and those were cases which could be easily differentiated by clinical symptoms and analytical methods.

Medical Section.

June 28, 1910.

Dr. J. MITCHELL BRUCE, President of the Section, in the Chair.

A Combination of the Auscultatory and Tactile Methods of Reading the Arterial Pressure (Systolic and Diastolic).

By GEORGE OLIVER, M.D.

(PRELIMINARY COMMUNICATION.)

IN making this brief communication I shall presume that you know something of the method of measuring the arterial pressure by means of the armlet and manometer. As you are aware, in applying this method we employ the finger for reading the systolic pressure (extinction of the radial pulse), and the eye for determining the diastolic pressure (the optimum oscillation of the indicator of the manometer). The object of this paper is to show that the reading of the pressures is rendered more definite and accurate by bringing the ear into play in conjunction with the finger.

Though the digital and visual methods have the advantage of simplicity, they are by no means free from defects. It is true that some observers regard their finger-tips and eyes as all-sufficient for this purpose; but such confidence is by no means generally entertained. It is easy to prove that the tactile capacity of a well-trained observer for deciding on the extinction of the radial pulse falls far short of actuality, by applying a wristlet connected with a compressed-air manometer containing a drop of spirit set at the pressure of from 90 mg. to 100 mg. when the spirit indicator will, as a rule, read the extinction or "still" point at least 10 mm. higher than the finger. In practice, too, the observer must often admit that there is an area representing several

millimetres on the scale in which the finger leaves him uncertain as to the actual arrest of the pulse. Besides the proved limitation of the tactile sense in determining the cessation of the circulation in the forearm, there are personal or accidental irregularities of that sense in different individuals which disqualify it for the purpose of deciding any unsettled points in connexion with the armlet method. Though some observers may think the finger is sufficiently accurate for the routine of clinical work, I am persuaded that the alliance of the ear with it renders the reading of the systolic arterial pressure much less uncertain in clinical observation than obtains with palpation of the pulse alone. Then, again, in determining the diastolic pressure, most observers find some difficulty in deciding on the optimum point of oscillation of the indicator; and in this case the stethoscope furnishes a much more definite reading of that pressure.

Having heard that the stethoscope had been employed somewhere in Germany and at Johns Hopkins University for the purpose of measuring blood-pressure with the armlet and manometer, I have lately been testing the efficiency of the auditory by the side of the tactile and visual methods. Some twenty-two years ago McKendrick wrote: "If a stethoscope be placed over a large artery, a murmur, sound, or bruit will be heard, caused by the blood rushing through the vessel narrowed by the pressure of the instrument."¹ Hence it is that when the armlet is brought to bear on the brachial artery, and the stethoscope is placed on the mesial line at the bend of the elbow, a throb is heard. In normal subjects the transition from silence to throb is quite definite, and, though varying in different cases, is fairly uniform in each individual, only rising and falling with alterations of the arterial pressure; and the throb begins low down on the scale—generally from 50 mm. to 80 mm., or perhaps 90 mm. When the armlet pressure is increased, the throb grows louder and maintains a higher degree of intensity for 20 mm. or so, and then gradually diminishes, and finally ceases as definitely as when it began. The cessation of the throb signifies the arrest of the circulation in the forearm, and therefore indicates the systolic pressure. It furnishes a very definite reading of that pressure, the difference of a millimetre or so in pressure forming the parting line between silence and the throb, or vice versa. Moreover, it provides a more sensitive reading of the pressure than the finger, being, as a rule, from 5 mm. to 10 mm. higher.²

¹ "Encyclopædia Britannica," 9th ed., 1888, xxiv, p. 105.

² On this point my observations confirm those of Fellner, *vide seq.*

There is a further matter worth mentioning in regard to the vanishing point. You know that the finger usually reads about 5 mm. lower pressure on the return of the pulse after its disappearance than on the obliteration of it by the upward pressure; but I have failed to detect this difference by the stethoscope when the pressure is slowly turned on and slowly withdrawn. I have found it quite safe to accept the upward reading, and this is certainly convenient.

The reading of the diastolic pressure is rendered definitely, and, I think, accurately, by halving the interval in which the throb is heard. For example, if the throb starts at 60 mm. and vanishes at 110 mm., the diastolic pressure will be 85 mm., being 25 mm. lower than the systolic pressure. This is about the normal difference between the pressures furnished by the stethoscope, and agrees with that afforded by the finger and the eye. I have frequently verified the correctness of this method of reading the diastolic pressure by observing its agreement with the point of maximum oscillation.

The auditory is a method quite readily learnt, and is easily applied. I suggest its adoption along with palpation of the pulse in the first instance, because you will learn for yourselves the points of advantage I have mentioned; and then it is not at all improbable that you will rely on it alone.¹ In order to make the method as sensitive and as practicable as possible, I have adopted a small resonating chamber (2 in. by $\frac{1}{2}$ in.) which is air-tight and fitted with two rubber tubes and ear-pieces. This tambour or resonator, mounted on a band and firmly buckled to the front of the elbow,² possesses certain advantages over a stethoscope, the end of which must be held *in situ*. Besides being more sensitive than a stethoscope, it liberates the fingers for the palpation of the pulse, while the ears listen to the throb; and it enables two observers to participate in the reading of the blood-pressure at the same time, as the resonator may be used with one ear only.

On looking up the literature of this subject, I find but one communication upon it, and that is a paper by Dr. Bruno Fellner, jun., of Franzensbad, read at the Twenty-fourth Congress of Internal Medicine, held at Wiesbaden in 1907. In that paper Dr. Fellner says he

¹ Since making this communication I have myself discarded the use of the finger in the routine use of the manometer.

² Since this paper was read I have been trying to simplify and improve the use of the tambour by making it a constituent part of the armlet, for I have found that the walls of the tambour in this position intercept the residual throb of the armlet, and the throb derived from the artery below the tambour is well pronounced. Mr. Hawksley, 357, Oxford Street, W., will supply the armlet fitted with the resonator and rubber tubes with ear-pieces.

had heard of the method having been practised by some Russian physician ; and, in the reported discussion on the paper, Herr Janowski stated that Korotkow, of St. Petersburg, proposed the method—grounded on his own experimental data—at the end of 1905, and that Krylow, of St. Petersburg, adopted it for clinical purposes. Janowski also referred to his assistant (Ettinger) as having employed the method for eight months prior to the meeting of the Congress in April, 1907.¹ Therefore, from this evidence, it would seem that we are indebted to Korotkow for suggesting the method. As I have not discovered that any British observer has either adopted it or published anything about it, I trust that I am justified in drawing your attention to it and in trying to arouse your interest in it. In a subsequent communication I hope to record the results of my observations with the method in different cases.

[Dr. Oliver demonstrated the auscultatory method, using a compressed-air mercurial manometer (recently devised by him and made by Mr. Hawksley), which possesses the following features: (1) Compensation for temperature variation secured by a vacuum enclosing the bulb ; (2) prevention of the spilling or loss of mercury by the adoption of a plug which permits free ingress and egress of air ; (3) compactness, yet withal a wide scale, measurements over 200 mm. in the horizontal position of the instrument being furnished by a second scale obtained by the weight of the mercurial column, plus compression of air ; (4) abolition of the momentum error of mercury ; (5) easy and more accurate reading of the scale in the horizontal and sideways positions.]

DISCUSSION.

The PRESIDENT (Dr. Mitchell Bruce), in thanking Dr. Oliver for his paper and demonstration, expressed his appreciation of the opportunity of seeing his latest invention. It was another instance of Dr. Oliver's ingenuity and zeal in increasing our knowledge of the methods of measuring the blood-pressure. From what he had had an opportunity of seeing, he believed that it provided a means of more easily and more correctly estimating what physicians were so anxious to estimate, the diastolic pressure.

Dr. ROLLESTON said that, as Dr. Oliver had kindly shown him the instrument and applied it on his arm, he could endorse what had been said about its accuracy in determining the diastolic pressure. A question which had since

¹ "Verhandlungen des Kongresses für innere Medizin," Wiesbaden, 1907, xxiv, pp. 404 to 407 and 415.

occurred to him was, what distinction could be drawn between the thud thus produced in normal subjects by the application of pressure and the thud audible by the stethoscope in the arteries in aortic regurgitation ?

Dr. OLIVER, in reply to Dr. Rolleston concerning aortic regurgitation, said he had not had any case of that disease so far, but he would anticipate that the thud which could be heard low down would gradually increase to the maximum point, and it would bring out a very low reading of the diastolic pressure; because one would have to go up for the systole, and probably commence from the area of zero rather than from 50 mm. or 80 mm.; so he thought the diastolic reading should come out very low for aortic regurgitation. The great point was that in using the stethoscope one naturally pressed on the artery with the end of the instrument, whereas in using the stethoscopic method with the armlet the bag was adjusted without pressure on the artery; therefore in aortic regurgitation there would probably be a short latent interval between the commencement of the observation and the appearance of the throb.

On Dorsal Percussion of the Thorax and of the Stomach, and a New Stomach Sign.

By WILLIAM EWART, M.D.

THE two objects of this communication, which is more specially concerned with the thoracic and upper abdominal organs, are closely related. (I) The first is a brief review of our present knowledge of the systematic *dorsal percussion* and plexigraphy of the viscera. This had hitherto remained incomplete, owing to the omission of the stomach. (II) The second is to fill that gap by introducing a method of "post-gastric percussion," and to describe a clinical sign in the back hitherto unpublished—"the dorsal gastric nucleus of resonance."

(I) BRIEF HISTORICAL REVIEW AND SUMMARY OF RESULTS.

(A) *The Early History of Dorsal Plexigraphy.*

Leopold Avenbrugger (1722-1798) had the inventor's divination of the pleximetric possibilities of his discovery. They are declared in the title of his work, "*Inventum Novum ex Percussione Thoracis humani ut signo abstrusos interni pectoris morbos detegendi*" (Vindobonæ, 1763; the first edition bears date of 1761). The following extracts define the extent to which he succeeded in their realization:—

§ III. 3^{to}.—"Sternum totum percussum resonat, ita clare, ac thoracis latera: excepto illo loco, cui cor pro parte subjacet; ibi enim paulo obscurior sonus percipitur."

4^{to}.—"Idem sonus per tractum spinæ dorsi observatur, quousque hæc concurrat ad efformandum thoracis cavum."

§ IX.—"Impera, cui dorsum percuties, ut ad anteriora se inclinet, humeros suos pectus versus adducat, gibbumque faciat, hæc eadem est ac prioris ratio, exactior scilicet sonitum evocandi methodus."

Scholium.—"Obvia hæc experimenta, quivis sanissimus in se, vel in aliis tentare potest: et inde capient omnes boni ex sonituum varietate fundamentum sufficiens, hocce signum in detegendis morbis pectoris non vilipendendi."

Corvisart (1755-1821), Avenbrugger's translator and commentator, did much; Laennec (1781-1826), the inventor of "mediate auscultation," did yet more for the clinical applications of the method. Piorry, to whom we owe the latest development, the method of *mediate percussion* and of *pleximetric percussion*, was the third inventor. But Piorry was also the originator of an entirely new method—that of *organic plexigraphy* or organography.

Dorsal Plexigraphy.—With the help of the pleximeter which he had invented, he was able to demonstrate the possibility of tracing at the surface of the body the outlines of subjacent structures, even when more or less distant from the surface percussed. His long life (Dec. 31, 1794 to 1879) was largely devoted to that work. But, owing perhaps to the excess of his zeal, and largely to his almost grotesque nomenclature, he earned for himself no better reward than the incredulity of his contemporaries and the oblivion of their immediate followers. He was the founder of a system, but not of a school. The pleximeter, which was to be revived in a more suitable form by the late Dr. Ernest Sanson, gradually fell into disuse, and plexigraphy as Piorry understood it, and with it also the solid results which he had secured, passed out of clinical existence for two entire generations of clinical teaching and study. The greatest loss involved in that suspended animation was the abeyance of the practice of dorsal plexigraphic percussion. His results in the dorsal regions, which we are now considering, will be briefly reviewed at another page.

In spite of unstinted labours, Piorry left dorsal percussion in an incomplete state, as may be gathered from his latest publication in 1866.¹ His descriptions related to some only of the structures for which

¹ "Traité de Plessimétrisme," Paris, 1866, cf. pp. 604, 619, and 673. The same illustrations also display Piorry's localization of the kidneys.

dorsal percussion is available—namely: (1) The vertebral column, (2) the heart, (3) the right, thicker end of the liver, (4) the kidneys, (5) the spleen, (6) the pancreas, and (7) the pelvic viscera. Moreover, complete accuracy was wanting. This was probably due to his having used the flat ivory pleximeter known under his name, which has much too broad a flange for any fine localization.

(B) *The Recent History, Before and Since the Introduction of Radioscopy.*

The history of dorsal percussion within our own time is easily summed up. It was neglected before the days of radioscopy under the impression that there was nothing worth percussing for in the back, and neglected since then under the impression that there was so much, as shown by radioscopy, that it was hopeless to percuss for it. To the credit of the clinical method be it said that these were impressions only, and that, although it may have passed almost unheeded, a true statement of the facts has been available for the profession. A more searching bibliographical inquiry may perhaps reveal the forgotten existence of some intervening instalments of research,¹ but, so far as known to me, the current practice of dorsal percussion in the closing years of the last century was still restricted to the search for either pleural or pulmonary resonance or dullness. Its finer uses for the delimitation of organs were in abeyance, and dorsal plexigraphy had not made any advance beyond the results published by Piorry. With these results I remained unacquainted until after the greater part of the work had been done which was published from time to time. The references are given in full, as they convey some general indication of the subjects dealt with:—

1889: "The Bronchi and Pulmonary Blood-vessels: their Anatomy and Nomenclature" (J. and A. Churchill, London, 1889).—1891: "On Accuracy in Cardiac Percussion" (*Lancet*, 1891, ii, p. 473).—1892: "Cardiac Outlines for Students and Practitioners" (Putnams' Sons, New York, 1892).—1896: "Practical Aids for the Diagnosis of Pericardial Effusion in Connexion with the Question as to Surgical Treatment" (*British Medical Journal*, 1896, i, p. 720).—1896: "Latent and Transient Pericardial Effusions" (*Lancet*, 1896, ii, p. 1446).—1896: "The Röntgen Rays and the Dorsal Examination of the Heart" (*Lancet*, 1896, ii, p. 1790).—1897: "Remarks on the Dorsal Test

¹ A. Cros, M.D. (of Paris), read a paper on "The General Principles of Pleximetric Organography" before the Medical Society of London in 1884 (cf. *Trans. Med. Soc.*, vi, p. 456); but his paper does not contain any reference to dorsal percussion.

for Pericardial Effusions: an Addendum" (*British Medical Journal*, 1897, i, p. 185).—1898: "On Latent and Ephemeral Pericardial Effusions" (*Transactions of Clinical Society*, 1898, xxxi, p. 97).—1898: "On Pleximetry and Pleximetric Bones and Viscera" (*Clinical Journal*, 1898, xi, p. 221).—1898: "Practical Points in Percussion and Auscultation" (*Clinical Journal*, 1898, xi, p. 221).—1898: "The Diagnostic Uses of Percussion of the Vertebral Spines, with General Remarks on 'Pleximetric Bones and Viscera,' with References" (*Lancet*, 1898, ii, p. 23).—1899: "Two Cases illustrating Gastric Dilatation Upwards and Backwards, its Relation to the Heart and Respiration, and its Treatment" (*Transactions of Clinical Society*, 1900, xxxiii, p. 202).

The practical results described in these various contributions were summed up in a paper read before the Medical Society of Cambridge on June 2, 1899,¹ together with a "map of dorsal percussion." Since that time I have not ceased to use the method and its data with great advantage. This renewed endeavour to raise dorsal percussion into its deserved position of acknowledged clinical usefulness was doomed to failure. The attempt again hung fire, and since then no additional contribution seems to have been made to medical literature descriptive of a systematic dorsal percussion of the mediastinal structures, of the heart and left auricle, of the liver, stomach, pancreas, and kidneys, or of *pericardial* and *peritoneal* effusions. Meanwhile, however, a definite actuality was given to dorsal percussion for differential diagnostic purposes by v. Korányi's observations in 1897 on pleural effusions in children, and in 1902 by Grocco's valuable test for free fluid in the pleura,² to which I shall presently refer. These are practically offshoots of the method of dorsal percussion in the clinical field of pathology.

But in the domain of *normal clinical anatomy* the first revival of the dorsal method was v. Korányi's praiseworthy attempt in 1906 to determine the percussion values of the entire series of spinous processes.³ This was an original investigation, as it was published whilst he was still in ignorance of the "dorsal map" of 1899, in which the individual characteristic percussion values special to each of the more important vertebræ are fully described. But it cannot be regarded as a progressive step, as it does not enter into the separate study of the individual vertebræ, and does not help us even up to the points demonstrable by the

¹ "On the Recognition of the Enlargements of the Left Auricle by Percussion, and on other Clinical Uses of Dorsal Percussion, including Percussion of the Pelvis," *Brit. Med. Journ.*, 1899, ii, p. 1167; and "On the Practical Aspects of Dorsal Percussion, and in particular of the Percussion of the Spine," *Lancet*, 1899, ii, p. 261.

² *Lavori d. Cong. di Med. int.*, 1902, Rome, 1903, xii, p. 190.

³ Loc. cit., cf. *Zeitschr. f. klin. Med.*, Berl., 1906, lx, pp. 295-313.

X-rays. Moreover, the outcome of the investigation, which was planned on the unfortunate principle of a "uniform" style of percussion to be applied to all the spinous processes, is not in agreement with the results which had been obtained by the analytical method, and the correctness of which is capable of verification. Instead of individualizing the percussion values, he was led to divide up the spinal series into five serial groups, each of which is described as possessing a different percussion value of its own. But even here I cannot quite agree with the estimation given of the regional sound-values. This fresh spinal mapping has, however, been adopted by Martin Nagel¹ in his important paper, and by others.

The latest actuality is a practical paper by J. C. da Costa, jun.,² also limited to spinal percussion, with special reference to its diagnostic uses for the study of pathological changes in the mediastinum. This again is based upon the "regional" spinal grouping of v. Korányi, and therefore misses some of the analytical advantage which might have been gained from the "vertebral" percussion map which was available.

This concludes the chronological history of the method of dorsal percussion. We shall now proceed to a brief critical review of its developments and of its present position.

(C) *The Results achieved in Dorsal Plexigraphy.*

There are practically four stages in its evolution which are easily studied in connexion with the four maps which are submitted to illustrate them: (I) Piorry's map; (II) the map of 1899; (III) Grocco's map; (IV) the present map (1910) or revised map of 1899.

(I) *Piorry's Map*.—As previously stated, Piorry's plexigraphic contribution was limited to the (1) spinal column; (2) the heart; (3) the liver; (4) the spleen; (5) the kidneys; (6) the pancreas; and (7) the pelvic viscera.

(1) *The Spine*.—It is superfluous to enter into the historical detail. That is dealt with in another paper communicated to the annual meeting of the British Medical Association, July, 1910, and exclusively devoted to the subject of "Vertebral Percussion in Diagnosis, with special reference to Surgical Diagnosis."³ The main fact is that he does

¹ "Der phys. Nachweis vergrößerter Bronchial-und-Mediastinaldrüsen," *Jahrb. f. Kinderheilk.*, Berlin, 1908, lxxviii, p. 46.

² "The Practical Value of Spinal Percussion for the Diagnosis of Diseases of the Mediastinum," *Amer. Journ. Med. Sci.*, Philad., 1909, cxxxviii, p. 815.

³ *Lancet*, 1910, ii, p. 470.

not seem to have realized the value of a separate study of the individual spinous processes, and that he deals with the spine as a whole. The *vertebral column* was easy to percuss. It is not, however, mentioned in the earlier of his works¹ (1828), but a full account of its percussion is given in one of his subsequent works² in 1837. Piorry percussed the spinal column as a separate structure; he did not make any attempt at a differential percussion of individual vertebræ.

(2) *The heart's* dorsal percussion³ is seen to be far from correct in its general outline. The post-cordial dullness is left in mid-air, as it were, both unsuspended and unsupported. There is neither any post-hepatic dullness beneath it nor any supra-cardiac dullness above it. Much less is there any outlining of that part of the heart which is

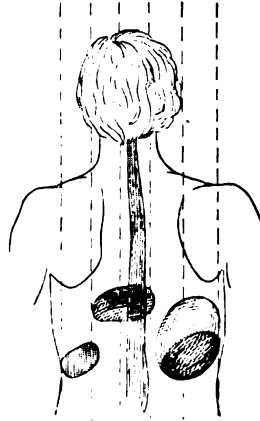


FIG. 1.

Piorry's dorsal percussion map, from the "*Traité de Plessimétrisme*," 1866, p. 248. (For his renal, pancreatic, pelvic and other percussion results, cf. pp. 604, 619 and 673.)

within the more direct reach of the pleximeter, the left auricle. It may be said of his description of post-cordial percussion that it is cursory, almost incidental, and not up to the standard of strict measurement or of anatomical localization.

(3) As regards *the liver*, Piorry's plexigraphy was both incomplete and inaccurate. The post-hepatic dullness which he depicts is limited

¹ "De la Percussion médiate et des Signes obtenus à l'aide de ce nouveau moyen d'exploration dans les Maladies des Organes thoraciques et abdominaux," par P. A. Piorry, Paris, 1828.

² "Traité de Diagnostic et de Séméiologie" (2nd ed., 1837), p. 558, No. 4501.

³ "Traité de Plessimétrisme et d'Organographie," &c., Paris, 1866, fig. 26, p. 248.

to the right third of the liver and rises much too high in the chest. Nevertheless, when after my own independent work in dorsal percussion I became acquainted with that of Piorry, I was so much impressed with his having noted an important point which I had missed, that I used the name "Piorry's nucleus" to identify a special and very obvious dullness, the peculiar features of which he had roughly indicated rather than described, and upon which is based an important method of hepatic diagnosis. This nucleus and the method will be referred to below.

(4) *A splenic dullness* is depicted by Piorry in the back. Normally, however, the splenic projection is more lateral than backwards. Indeed, unless pathologically enlarged, the spleen does not give any important dullness in the back in many subjects.

(5) *The kidneys* were not quite accurately percussed, and their dullness does not correspond to their exact anatomical position.

(6) *The pancreatic dullness*, as described and sketched, likewise in the "Traité de Plessimétrisme," 1866, is open to the same serious criticism.

(7) *The lower dorsal and pelvic* percussions of Piorry need not be described beyond a passing reference, as they lie beyond the scope of this paper.

Lastly, attention should be called to Piorry's remarkable "Atlas de Plessimétrisme," published in 1851 as Tome ix of the "Traité de Médecine et de Pathologie," Paris, 1851.

(II) *The Map of 1899*.—The map and some description of its details will be found in the papers published in the *Lancet* and *British Medical Journal*.¹ We may profitably postpone any remarks upon it, as it will be discussed presently in connexion with its revised edition (1910).

(III) *Grocco's Map*.—This possesses, in spite of its relating exclusively to the clinical diagnosis of free pleural effusion, an important historical bearing upon the method of dorsal percussion, and some collateral bearing, too, upon spinal percussion; although very little, if any, stress seems to have been laid either by v. Korányi or Grocco, or by subsequent writers, upon the fundamental fact that the spinal note itself is modified, and that its modifications are a most valuable confirmation of the genuineness of the triangular dullness. It also throws considerable light upon the mechanism of production of the latter.

The literature relating to Grocco's sign is too extensive to be dealt with exhaustively in this communication. It will suffice to enumerate the chief *practical developments* of the subject which have been referred

¹ Loc. cit.

to in our own medical literature, as the chief theoretical aspects have recently been considered in a "Note on Some Theories as to the Mode of Production of Grocco's Paravertebral Triangle of Dullness."¹

(A) *As regards the technique* of Grocco's original method, which needs no description here, an important modification was introduced in the first critical account of it published in the English language.² That paper describes a procedure which acts as an efficient check upon the genuineness of the sign quâ sign of *free* fluid within the pleura, to the exclusion of (1) any spurious findings, as when a triangle of dullness may be outlined, though in reality this is only an unconscious artefact of percussion, and (2) also of any genuine dullnesses more or less closely resembling in their configuration a true Grocco's triangle, and yet due to some totally different origin (thickening, consolidation, tumour, &c.).

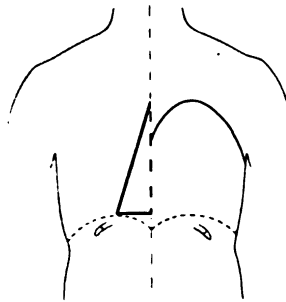


FIG. 2.

The paravertebral triangle of dullness (Grocco) in free pleural effusion.

The value of the sign for diagnosis depends entirely upon the absolute elimination of these elements of confusion.

The *test and counter-test*³ carried out by varying the posture, and in particular by an alternate percussion in the right and in the left decubitus, may therefore be regarded as an essential part of the technique, without which the sign cannot carry conviction.

Another practical point is the confirmatory value of a skilled percussion of the spinous processes themselves, as their behaviour under cross-examination disposes of the fundamental question as to an alleged derivation of the genuine triangular dullness from the normal paravertebral muscular masses.

¹ *Lancet*, 1909, i, p. 1738.

² "On the Value of Grocco's Paravertebral Triangle as a Physical Sign for the Diagnosis of Pleural Effusions," *Lancet*, 1905, ii, p. 216; *cf.* also p. 316.

³ *cf.* *Loc. cit.* and *Lancet*, 1907, ii, p. 49.

I have also demonstrated that in *double* pleural effusion the sign is readily obtained by percussion in spite of the presence of fluid in the other pleura. In those cases, therefore, there are *two reciprocal triangles*; but, like the effusions themselves, they are seldom of the same height.

(B) *As regards the range of Grocco's sign*, evidence was adduced in the same paper and in subsequent communications that, although

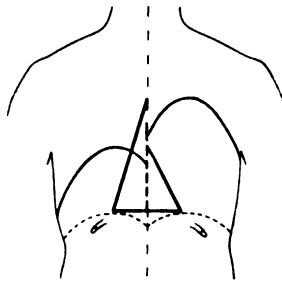


FIG. 3.

The reciprocal triangles, usually of unequal size, obtainable by percussion in double pleural effusion. The dullness of the spine itself is greater in the small triangle than above it, because under the influence of both effusions.

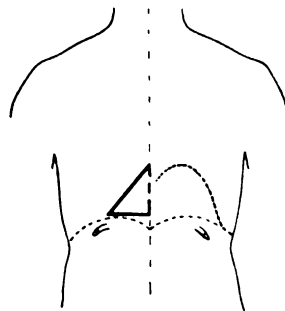


FIG. 4.

Grocco's triangle, due to a paraspinal abscess raising the right wing of the diaphragm.

always due to fluid, the production of the genuine paravertebral triangle is by no means limited to fluid within the pleura, but may also be due to fluid accumulations beneath the diaphragm, and in particular to (1) *unilateral* fluid collections, such as an abscess,¹ and to (2) *bilateral* fluid collections, such as the intra-peritoneal.

¹ *Lancet*, 1905, ii, p. 216.

*The bilateral isosceles triangle, with broad base, is therefore a further sign, though often a superfluous one, added to our list of the physical signs of ascites.*¹ Thus the paravertebral triangle is made available as a *confirmatory test* for ascites when this is of sufficient extent to raise the diaphragm slightly.

The Pelvic Test for Ascites.—This is also, perhaps, the best place to introduce another test which is a *practical fine-diagnosis test* for ascites. It is always available in doubtful cases, as in them the free application of postural tests is not precluded by any great encumbrance with fluid. This test has no connexion whatever with Grocco's method, but belongs simply to the method of dorsal and spinal percussion. The best name for it would probably be the "pelvic test." It is intended for the detection of ascites in its earliest stages. It consists in causing the patient to alternately sit up and lie prone with slightly raised pelvis.

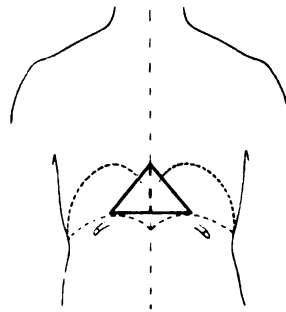


FIG. 5.

Diagram illustrating the bilateral triangle, with broad base, obtained in considerable ascites raising the diaphragm.

Our percussion of the lower lumbar or of the sacral vertebræ will then yield *alternately a clear resonance and a water-level dullness*, and will enable us to identify the presence of very small quantities of fluid not otherwise demonstrable. I have found it most valuable for the recognition of the early effusions connected with hepatic disease.

(IV) *The "Dorsal Map" of 1899 Revised (1910).*—The faults of the old map are partly due to bad drawing, partly to imperfect accuracy in the results of percussion, and partly to omissions, all of which are now, so far as possible, corrected.

(A) *The Vertebral Column and the Spinous Processes.*—Here there is hardly any need for rectification. Moreover, we shall be spared

¹ *Lancet*, 1907, ii, p. 49; 1909, i, p. 1739.

the necessity for any lengthy details, as the whole subject of spinal percussion is dealt with in the paper "On Percussion of the Vertebræ," communicated to the annual meeting of the British Medical Association, 1910. Attention is specially called to the essential distinction between the purely pleximetric "vertebral" note, special to the spinous process itself, and the *mixed* note, special to "rachidian" percussion, in which the pleximetric conduction from the vertebra is not directly continued to the actual surface, but only approaches it via the transverse processes, which are themselves clothed with muscles. As conducted through them the vertebral pleximetry is *attenuated*, and it is also *impure*.

(A¹) *The Columnar or Rachidian Percussion*.—This is mainly a non-pleximetric dullness, as we are not hard down upon the bone. "Dullness"

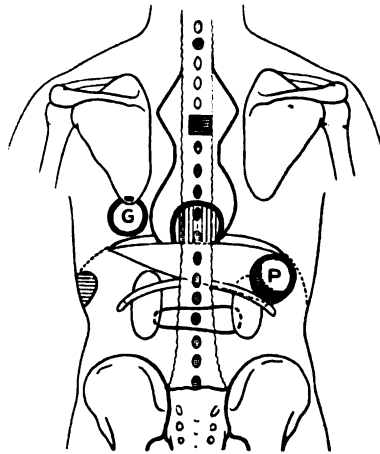


FIG. 6.

The complete map of dorsal percussion, including the "suprahepatic" partial dullness, "Piorry's nucleus" of hepatic dullness, and the "gastric nucleus" of resonance.

is a relative expression. The broad and continuous "spinal" band of Piorry, or the two "paraspinal" bands which I have described, have a varying element of resonance according as we use light or heavy percussion. But their "note of differentiation" is one of *subresonance*, or call it one of *subresonant dullness*. Searching percussion may also differentiate an outer band corresponding to the muscular masses. This presented formerly no practical interest; but some interest now attaches to it in its lower part, where it broadens out into the lumbar muscles dullness, because of its having been credited by Forbes-Ross, Roch and Dufour, and others, with the production of Grocco's triangular dullness, of which it is demonstrably innocent.

The individual percussion notes of the normal vertebræ (without entering into finer analysis) are as follows: First to sixth cervical, subresonant dullness; seventh cervical and first dorsal, less resonant than the above; second to fourth dorsal, definitely *resonant*; fifth dorsal ("infratracheal") definitely *dull*; sixth dorsal, subresonant; seventh and eighth dorsal ("upper cardiac"), less resonant than the sixth; ninth dorsal ("auricular"), *dullness*; tenth dorsal ("cardio-hepatic"), subresonant dullness; eleventh dorsal ("hepatic"), duller than the tenth; twelfth dorsal ("hepatic"), *duller* than the eleventh; first lumbar, subresonant dullness; second lumbar ("pancreatic"), definitely *dull*; third to fifth lumbar ("intestinal"), variously resonant or subresonant, with a tendency to dullness in very muscular or fat subjects; sacral, varying resonance; coccygeal, resonance, subresonance, or dullness.

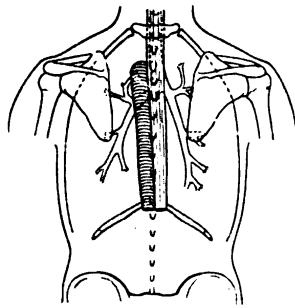


FIG. 7B.

Diagram of the fifth dorsal spine and of the asymmetrical position of the œsophagus and trachea.

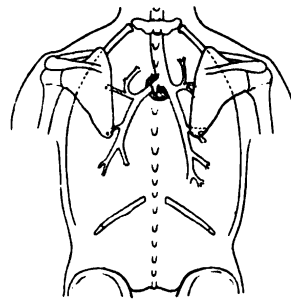


FIG. 7C.

Diagram of the fifth dorsal spine in its relation to the bifurcation and to the infra-tracheal gland.

The prominent features in the series are: (1) The isolated dullness of the fifth, of the ninth, of the twelfth dorsal and of the second lumbar spines, and the associated existence of a small "infratracheal dull patch," of a "left auricular dullness," and of a "pancreatic dullness." (2) The isolated resonance of the second to the fourth dorsal, of the sacrum and of the coccyx, and in lean subjects of the third to fifth lumbar. (3) The isolated subresonance of the sixth dorsal, of the seventh and eighth dorsal, and of the tenth dorsal ("cardio-hepatic" vertebra); and (4) the mitigated dullness of the eleventh and twelfth dorsal ("hepatic" vertebræ). (5) The "lozenge-shaped upper interscapular dullness," the "supra-cardiac interscapular dullness," the true "post-cordial dullness," and the "pancreatic dullness," all of them *relatively*,

not absolutely, dull, are of great clinical importance. (6) The "lower dorsal dull patch" is grafted pathologically in pericardial effusion upon the central hepatic dullness; normally it is sometimes to be traced in small children as a dulling influence conveyed from the heart itself. The "paraspinal bands of subresonant dullness" extend the whole way.

The diagnostic applications to be derived from a knowledge of these normal conditions are varied and important. Their description, which would claim too much space, will be found in the papers mentioned above (1899).

(B) *The Dorsal Visceral Percussions, "Non-Pleximetric," or by ordinary Thoracic Conduction.*—(2) *The Interscapular Dullness.*—As in all our dorsal visceral percussions, we are dealing here with ordinary, non-

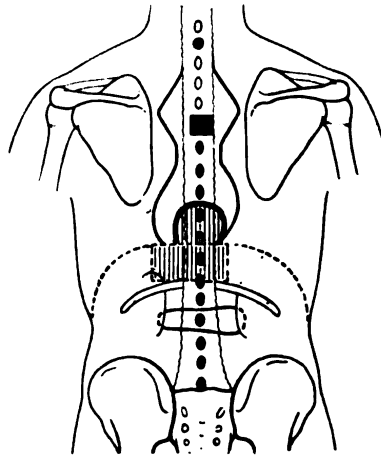


FIG. 8.

The objects for percussion down the length of the spine: (1) The lozenge-shaped upper interscapular dullness; (2) the infra-tracheal dull patch; (3) the supra-cardiac dullness; (4) the post-cordial dullness and the left auricular dullness; (5) the lower dorsal dull patch of the liver; and (6) the pancreatic dullness.

pleximetric values. If the stroke is so heavy as to elicit the pleximetric pulmonary note special to the ribs, we are lost. Its more accurate description needs further study, for which I have not had leisure. But there is no major modification to note in the original description of this most important dullness as a lozenge-shaped, slightly subresonant dullness, compounded from the solid elements of the upper mediastinum, of the pulmonary roots, and of the great vessels. It expands again below into the supra-cardiac and into the cardiac dullness proper.

(2a) *The Diagnostic Applications.*—This, in the field of dorsal thoracic percussion, is by far the most important region for our clinical purposes, both medical and surgical, because of the variety and importance of the affections localized into it, and because most of them (including aneurysm, cancer, and glandular affections) are not accessible for identification from the front. For their consideration I must again refer to the papers published in 1899.

(3) *The Cardiac Dullness.*—The old description, so far as it goes, still serves, and there is little fresh to add to it except two mere details in the cardio-hepatic boundary. (a) My percussions of recent years have brought out more clearly the retiring angle normally present at both sides at the base of the post-cordial dullness, between the auricles and

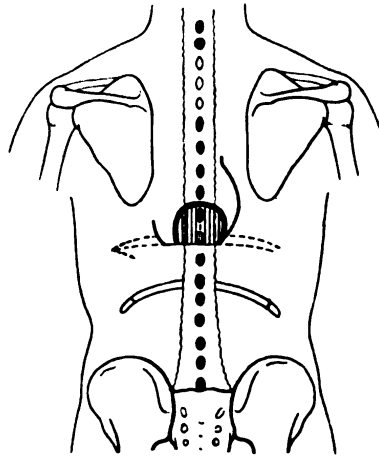


FIG. 9.

The dullness of the auricles and of the left ventricle in their relation to the hepatic and supra-hepatic lines.

the liver. This feature is well borne out by the X-rays examination, and is perceptible in any good skiagram. (b) The lower boundary is also slightly modified by the introduction of a "supra-hepatic" line not previously described. (c) The left auricular dullness was correctly described eleven years ago; also the curved outline of the right auricle, resembling the outline of the same auricle when percussed in front between the parasternal line and the infrasternal notch.

(3a) *The Diagnostic Applications.*—The confirmatory value of the post-cordial outline needs no emphasizing. It is of special help, as originally pointed out in 1899, in connexion with enlargements of the

left auricle, as their range is chiefly in the back. The right auricle enlarges chiefly in the transverse diameter, towards the right axilla, whilst it almost spans, as the left auricle actually does, the entire depth of the chest—viz., from the vicinity of the ninth vertebra to the third space. The *pericardium*, which possesses no dullness of its own, acquires one when charged with fluid. The tendency of the latter is, for anatomical reasons connected with the *venæ cavæ*, to depress the anterior rather than the posterior part of the diaphragm and liver. The tenth vertebral level is maintained by the central tendon, but the two angles of resonance just now referred to will be merged into the post-cordial dullness if there be sufficient fluid present. The only conclusive dorsal test for pericardial fluid is to be found below the level of the latter. The dullness of the fluid itself is imperfectly transmitted horizontally to the back; but its dulling influence on the subjacent portion of the liver is transmitted in the shape of the well-known “lower dorsal dull patch” to the “hepatic vertebræ,” and to a quadrilateral patch the right and the left section of which are of unequal size, in agreement with the asymmetrical insertions of the pericardium.

(4) *The Hepatic Dullness*.—The only corrections needed are in the direction of greater accuracy. They relate to (a) the upper boundary of the dullness; and (b) the features of *Piorry's nucleus*.

(a) *The Supra-hepatic Line*.—I had not formerly realized that a complete delineation of the liver is almost as easily got behind as it is in front. Its upper level, that of the tangential plane, or “supra-hepatic” line, is now entered in my tracings in the back as well as in front. And, as shown in the paired superposed anterior and posterior tracings I submitted to you, the two lines agree in their level.

(b) “*Piorry's Nucleus*” of Dullness is, as I originally described, an almost circular dull patch at the right outer pulmonary base, being in reality the dullest part of the thick end of the liver at its thickest. It therefore coincides with the extreme fundus of the retort-shaped post-hepatic dullness. The more the fundus enlarges, the larger will the nucleus be; its extension taking place chiefly upwards and outwards, but also downwards. As similar directions are also followed by the enlargement of the fundus in front, the anterior and the posterior dullness ultimately merge into one dullness, as explained in the same papers (1899).

In health *Piorry's nucleus* is an almost constant quantity in size and in position. Its adult male diameter is about $2\frac{3}{4}$ in., and its position is such that it just touches the outer part of the summit of the right kidney

dullness. This gave me the key to the mechanism of the constant and peculiar conformation of the nucleus. The circular nucleus of dullness is in reality made up of two segments possessing different percussion values—namely, (a) a rather smaller inferior and inner segment of crescentic shape, and (b) a larger segment completing the area of the circle. This larger outer and upper segment is *subresonant*, *the crescent itself is dull*. In Piorry's map the size of the nucleus is exaggerated out of all proportion; and the remainder of the dorsal hepatic dullness is not depicted and does not appear to have been made out by him. Moreover, his nucleus is oval instead of round, and its duller inferior segment is not crescentic, nor is it continued up towards the spine. It presents an historical interest only. The causation of this normal and invariable

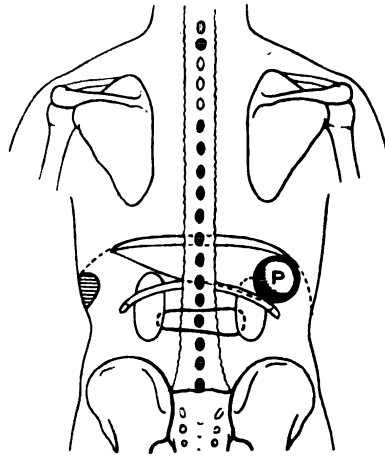


FIG. 10.

The dorsal percussion of the abdominal organs. The liver and Piorry's nucleus of hepatic dullness. The crescentic absolute dullness within the nucleus is shown to be due to dulling contact with the right kidney.

arrangement had remained a mystery to me until I realized that the dullness of the crescent was due to the damping effect of the renal contact with the twelfth rib and with the liver itself. The remainder of the nucleus owes its relative resonance to the sonorous influence of the pulmonary tissue overlying the upper and outer surfaces of the liver at the axillary base. This is, in short, another instance of the same muting of vibrations which we have already recognized in the left lobe of the liver as the cause of the lower dorsal dull patch of pericardial effusion, and also in the spinal column as the cause of the paravertebral triangle of dullness (Grocco) from pleural effusion.

It will be noticed that a normal Piorry's nucleus has its upper boundary at the hepatic line, not at the supra-hepatic line, and that it extends downwards to the level of the lower border of the liver.

(4a) *The Diagnostic Applications.*—Piorry's nucleus is a great resource as a finder for the twelfth rib and for the right kidney, which it touches, and for the lower hepatic border and the whole dullness. The main "hepatic line" having been found, the supra-hepatic line is more easily determined and the entire liver can be percussed out. The value of the *complete dorsal* percussion of the liver can hardly be overestimated. It is our touchstone for the diagnosis and localization of hydatid, of abscess and other hepatic disfigurements. In every-day cases it is the only reliable test for the actual size of the liver. For, if percussed only anteriorly, the liver might be judged to be considerably enlarged when it was merely tilted forwards, a type which we may term "the apron liver"; or, on the contrary, to be considerably diminished when only levered up by the abdominal overfullness, as "the capsized liver." *Genuine enlargement* is unerringly identified not only by the raised upper level of the dorsal supra-hepatic and hepatic lines, associated with some corresponding increase in the size of Piorry's nucleus, but, above all, by their progressive extension towards the axilla, where, in cases of considerable enlargement, they join the anterior supra-hepatic and hepatic lines and define a continuous hepatic dullness circling round the entire right side (*cf.* fig. 12). Lastly, it is superfluous to dwell again upon the satisfactory confirmation afforded by the demonstration that the quadrilateral *lower dorsal dull patch* in pericardial effusion is nothing more than the sub-pericardial segment of the hepatic dullness locally intensified and mathematically delimited by the floor of the waterlogged pericardial space.

(5) *The Splenic Dullness.*—A much enlarged spleen must always encroach upon the dorsal map. A normal splenic dullness cannot, however, be always traced beyond the axillary base; certainly not in the position and shape depicted by Piorry. This is essentially a question of width of the thorax and of inflation of the lung. In subjects with a conformation specially favouring its detection the small element of dorsal splenic dullness would be readily recognized, as stated in 1899. Its continuity with the axillary splenic dullness and its close relation to the eleventh rib and to the left renal dullness are reliable guides.

(5a) *The Diagnostic Applications* are obvious and important, and need not delay us

(6) *The Renal Dullness*.—In this respect the map of 1899 does not call for any material corrections, but Piorry's percussion was not precisely correct.

(6a) *The Diagnostic Applications*.—The definiteness and accessibility of the field of renal percussion furnish the physician, and yet more the surgeon, with opportunities which have not hitherto been utilized to their full extent. It is something to obtain a demonstration of the presence of the two kidneys in their proper place. More often, however, the point at issue is the question as to any alteration in their size or as to the presence of any inflammatory or neoplastic deposit; and on these points a skilled percussion can throw considerable light. We cannot subscribe to Piorry's too ambitious belief in the possibility of mapping out the suprarenal capsule. But a suprarenal tumour of any considerable size would undoubtedly modify the outline of the renal dullness in a recognizable degree, although our percussion could not afford proof of the localization in the capsule rather than in the kidney.

(7) *The Pancreatic and Visceral or Mesenteric Dullness*.—These dullnesses were correctly identified in 1899 with the dullness of the *second lumbar spine*, and that connexion was demonstrated by the study of a case originally suspected of latent carcinoma of the pylorus, but which proved on subsequent laparotomy to be one of chronic gastric ulcer. The normal second lumbar dullness is our working basis in the clinical examination for abnormal dullnesses at the level of the first two lumbar spines, which may perhaps be termed "the mesenteric spines."

The Pancreatic Dullness.—In the case referred to, the discovery of a patch of pronounced dullness to the right of the second lumbar spine (*cf.* fig. 7A) led me to suspect the possibility of disease of the head of the pancreas instead of pyloric carcinoma. The operation, from which the patient completely recovered, disproved both these diagnoses. The pancreas was free from disease, but its normal dullness had been intensified by inflammatory thickening around it. Previous to that date I had not attempted the dorsal percussion of the normal pancreas. It is only comparatively recently that I have turned my attention to that study. I now find that it is, as a rule, possible to trace the normal pancreatic dullness in the shape of an oblong horizontal patch, extending to the right and to the left of the second lumbar vertebra, and overlapping the two kidneys, as shown in the diagram. I now practise pancreatic percussion as part of the systematic examination of the body.

(7a) *The Diagnostic Applications*.—The region in question compares in its importance for diagnosis with the interscapular region. It is the

abdominal region *par excellence* for dorsal percussion, as a satisfactory anterior percussion of it is always, and a satisfactory anterior palpation usually, impossible. It is also *par excellence* the region of the unknown, in respect of the great variety of the possible complications. It is unnecessary to dwell at greater length upon that very large subject.

(8) *The Dorsal Pelvic Percussions.*—Under this heading *vertebral* percussion is represented by *sacral and coccygeal* percussion, and *pelvic* percussion by that of the other pelvic bones. The account which had been given of them in 1899 calls for no amendment, and may be consulted in the papers previously referred to.

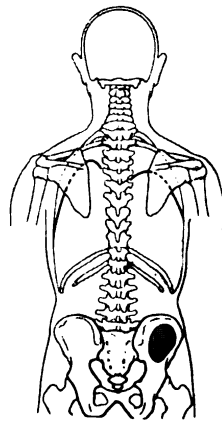


FIG. 10A.

Dorsal percussion of the ilium for abscess, appendicitis, &c. This also shows the normal difference in level between the apex of the right lower lobe and that of the left.

(8a) *The Diagnostic Applications* are sufficiently obvious. There could hardly be a more striking illustration of their use than the instance quoted in one of the papers. A pelvic abscess, which had given rise to a clearly defined dullness of part of the right ilium, failed to be discovered by a subsequent laparotomy. But it was found at the post-mortem examination a few days later, and proved to be a fæcal abscess bound down by adhesions behind the cæcum. It was presumably the late result of an appendicitis which had been overlooked. Recent experience convinces me that this mode of examination is of practical value for the identification of the disease in its early stages.

This concludes the up-to-date review of the "dorsal" percussion map, with one exception, however, which we will now consider—that of the missing gastric percussion.

(II) THE DORSAL PERCUSSION OF THE STOMACH. THE NORMAL AND PATHOLOGICAL POST-GASTRIC RESONANCE, AND THE "DORSAL GASTRIC NUCLEUS OF RESONANCE."

I am unfortunately unable to contribute an exhaustive bibliographical account of the subject, and to say whether any systematic use of dorsal percussion for descriptive and for clinical purposes may have been reported by others, or how long the opportunities afforded by the X-rays, and more recently by the orthodiascope, may have been utilized for a dorsal examination of the stomach. I may, however, refer to a paper on "Two Cases Illustrating Gastric Dilatation Upwards and Backwards, its Relation to the Heart and Respiration, and its Treatment."¹ In that paper clinical evidence was adduced to show that the anterior gastric resonance, normally limited to Traube's area, might in given cases be so greatly increased that its extension into the axilla and into the back would encroach upon the posterior basic area of pulmonary resonance. The upward distension of the stomach giving rise to this increase sets up various degrees of cardiac oppression or cardiac distress. Since that date, more even than before, cases of this kind have been frequently coming under my notice at a late period of the symptoms, their gastric causation not having been realized. This suggests the inference that widespread recognition has not yet been awarded to the fact that evidence of upward gastric dilatation may often be obtained by an examination in the back, as well as by an anterior percussion.

The other aspect of the subject, that of the possible occurrence of a non-pathological post-gastric resonance, had not, however, come to my mind before the year 1909, when I realized the existence of a normally manifest and constant "post-gastric nucleus of resonance," definite and uniform in its situation and size, which all my dorsal percussions had previously overlooked. Its percussion has since then proved to me of genuine clinical service.

The Normal Gastric Nucleus of Resonance.—The data as to situation, shape, size, and tone-value are simple.

(1) *Situation.*—The gastric nucleus is situated immediately below the inferior angle of the left scapula, which it may or may not faintly overlap. This is our rough guide to its identification. *The accurate localization* of its boundaries, which are as true as those of the post-cordial dullness, is much facilitated by the visceral surface markings

¹ *Trans. Clin. Soc. Lond.*, 1900, xxxiii., p. 202.

which have already been established. The wonder is that its existence should have so long escaped notice. This is the more remarkable as the self-same circular patch had been delineated by myself as early as 1896,¹ in an entirely different connexion, as defining in some cases of pericardial effusion an auscultatory area of tubular breathing and ægophony.² I had overlooked the more important normal feature attaching to its percussion. The precise localization is given by the dorsal markings of the cardiac and of the hepatic dullnesses. The circle of the nucleus is inscribed between the left post-cordial boundary, which it does not quite touch, and the supra-hepatic line. It is therefore possible to draw its outline on the back before having percussed for it, by utilizing also the rough guide of the scapular angle, provided the two lines in question have been correctly determined. I have not found any alteration in the situation from varying the decubitus.

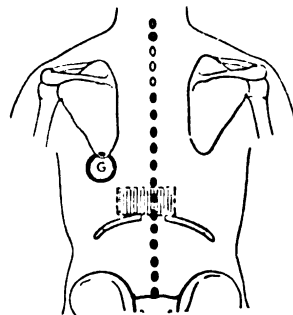


FIG. 11.

The gastric nucleus of resonance and the "lower dorsal dull patch"

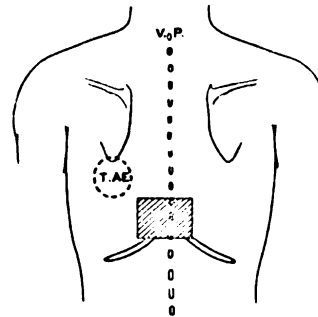


FIG. 11A.

The nucleus of tubular breathing and ægophony (1896) and the "lower dorsal dull patch" in pericardial effusion.

(2) *The shape* of the normal nucleus is invariably almost circular.

(3) *The size* varies with that of the individual. It is, however, by no means proportional to the body height. In children it is of course smaller in absolute measurement, but at the same time it is apt to be relatively larger than in the adult, and may perhaps prove on further investigation to be more prone to fluctuations in size in the same

¹ *Brit. Med. Journ.*, 1896, i, p. 720.

² These pressure signs have attracted the attention of various clinical observers (Lins, Reiss, Bamberger, &c.). The latter has localized them to a region closely corresponding to that here described.

individual. In the adult the average nucleus measures from 2 in. to $2\frac{1}{2}$ in. in diameter.

(4) *The percussion note* is one of increased resonance and of *tympanitic* quality, using the term in its original sense of drum-like *hollowness*. The nucleus is therefore more resonant than any of the surrounding surfaces. Is this merely a contrast effect due to the fact that below it and to the right of it there are duller surfaces, those influenced by the liver and by the heart? Or is there an actual positive excess of resonance over and above that of the lung itself? The correctness of the latter view is proved by two facts: (a) the boundary of the special resonance does not run with the boundaries of the special dullnesses, which indeed it does not always absolutely touch, but it runs in a circle; moreover (b) the boundary is equally traceable on the outer side where there is no element of dullness, as the inconstant dorsal dullness of the spleen is situated at a lower level. We may conclude, therefore, that the resonance of the normal nucleus is a genuine positive resonance due to some sonorous influence other than that of the lung itself.

The Causation of the Resonance.—Thanks to the X-rays, and in particular to the orthodiagraph, the clinical value of which has recently been brought to the notice of the Section by Dr. W. Pasteur, we are now in a position to frame some provisional inferences as to the mechanism of production of this resonance. In the first place, we must hold to the fact that the nucleus of resonance is *intrathoracic*, not intra-abdominal. It is situated above the hepatic line, which gives us one of the levels of the diaphragm. It is therefore not a horizontal projection backwards of the stomach-balloon (Magenblase). In the second place, the normal stomach does not rise quite up to its lower level. We must remember that the left lobe of the liver normally intervenes between the stomach and the diaphragm. Therefore, if this resonance be in reality due to the stomach, it must have been transmitted upwards by solid conduction through the liver. Lastly, the orthodiascope demonstrates to us that the air space of the normal stomach-balloon is exactly subjacent to the nucleus. There seems, therefore, to be some ground for a provisional inference that the nucleus is the result of the deep-seated resonance of the stomach, and for the possible inference that the striking constancy in the position and in the circular shape of the nucleus may be determined by some kind of "echo" effect, or perhaps rather of "sound-refraction," the liver acting as a lens for the resonant waves.

THE CLINICAL SIGN AND ITS USES.

I must not delay you with more than a mention of the *anatomical or descriptive value of the sign*. The gastric nucleus satisfactorily completes the dorsal map to the advantage of the teacher and of the student. Let me only allude briefly to the analogy and to the differences between our two nuclei: (1) Their situation at the right and at the left thoracic base respectively, their shape and their size, are almost symmetrical. (2) But in their relation to the supra-hepatic line they are essentially asymmetrical. Piorry's nucleus—the nucleus of hepatic dullness—is below the supra-hepatic line, and below the diaphragm; it is intra-abdominal. The gastric nucleus, the nucleus of stomach resonance, is above the supra-hepatic line and above the diaphragm; it is intrathoracic. (3) In addition to the contrast between the dullness of the one and the resonance of the other, there is the further contrast that the resonance of the gastric nucleus is *homogeneous*; whilst the dullness of Piorry's nucleus is of a composite or "magpie" description.

The technique of the new sign is most simple and rapid. Thanks to the rough guide of the lower scapular angle we may, for the purpose of mere confirmation in all those cases where there is obvious stomach soundness, dispense with the more thorough technique which implies, as a preliminary, a careful percussion of the post-cordial and of the post-hepatic dullness. We may, indeed, trace our gastric nucleus beforehand and apply ourselves at once to its verification by accurate delimitation.

It may be a comfort to many that the preliminary *identification* can be carried out without the use of any pleximeter. As the pleximeter is a special means of identifying slight dullnesses by accentuating them, there is no particular indication for its use in searching for a resonance. For this reason it is best to give two or three strokes of finger-to-finger percussion as an introduction to the finer work of *linear delimitation*. For this accurate delimitation, however, unaided digital percussion is much too difficult for the average observer. It can only succeed in the hands of the skilled expert. The only easy and safe method is to use a pleximeter provided with a small flange—Sansom's pleximeter is by far the best—and no hammer, but the middle finger as a percussor. Any beginner availing himself of this help can quickly learn to trace out with fair accuracy any boundaries of dullness or of resonance previously identified, and therefore, in this instance, the circular boundary of the gastric nucleus.

THE CLINICAL VALUE OF THE SIGN.

For clearness it is necessary to divide this subject into two sections. Here again we shall find striking analogies with the observations relating to the liver and to Piorry's nucleus, and some essential differences, particularly in regard to malpositions. Malpositions of the liver are most commonly only due to tilting, either forwards or backwards, and there is no such thing as an ephemeral distension of the liver. True ptosis again is not very common. The stomach, on the other hand, is very liable not only to distension and *dilatation*—and this may take place chiefly downwards or chiefly upwards, chiefly forwards or chiefly backwards—but also to *ptosis*; and, moreover, the two sets of change are apt to occur in *combination*. The first object in our complicated inquiry is to ascertain that none of them are present in any major degree. These preliminary remarks define the two main sections of our analysis of the subject of dorsal gastric percussion: (A) *The variations in size of a gastric nucleus* of normal situation and outline; and (B) *the abnormal extensions of the dorsal gastric resonance* either backwards or forwards towards the spine, or forwards into the axilla. These are the abnormal *dorsal gastric resonances with deformation* of the gastric nucleus.

(A) *The Variations in Size of the Gastric Nucleus.*—(a) The rapid identification of a normal gastric nucleus, normal in shape, in size, and in situation, is of great practical value in our routine clinical examinations. It supplies us in the briefest space of time with a probability that *the major dorsal gastric abnormalities are absent*. (b) The discovery that the nucleus, although normal in shape and in situation, was larger than normal, would raise a suspicion as to the existence of some other variety of gastric abnormality calling for further examination. At the same time it might suggest a probability that the trouble was only slight, and perhaps merely functional. The next step would be to compare notes with the results of our percussion of Traube's area, which would probably be found to be also enlarged. This would provide a striking parallel with the simple enlargements of our Piorry's nucleus in the liver. (c) As a clinical fact it not infrequently occurs, as shown in some of the tracings exhibited, that Traube's area may be considerably increased in size, and that its resonance may be continued over a considerable surface of the epigastrium, in spite of the absence of any notable increase in the diameter of the dorsal nucleus.

(B) *The Deformations of the Gastric Nucleus.*—The loss of a perfect circular outline is the first step towards the disappearance of the gastric

nucleus. Just as Piorry's nucleus was shown in 1899 to be involved in gradual extensions of the hepatic dullness towards and into the axilla, precisely in the same way the gastric nucleus may be the starting point of progressive encroachments outwards of an enlarging area of dorsal gastric resonance. This is the special feature of the more important variety, not, I believe, previously definitely described, of the two types of upward dilatation—namely, the *dorsal* or *backward type of upward dilatation of the stomach*. My paper in the *Clinical Society's Transactions* (vol. xxxiii), mentions this type in the title only, but does not specify the essential difference between the two distinct clinical conditions. The existence of a mainly *dorsal dilatation, which attacks the heart from behind*, instead of attacking it from the front, as in the case of the anterior upward dilatation, is in itself a sufficient excuse for this advocacy of a dorsal examination of the stomach which I had not previously urged. It implies that a determination of the dorsal gastric resonance is not a refinement or a luxury, but a duty which cannot be neglected without serious risk to our diagnosis and to our treatment of one of the most severe and dangerous forms of heart distress of mechanical gastric origin.

The Various Types of Upward Dilatation of the Stomach as revealed by Percussion.—It will have been gathered from the above that a gastric examination by percussion consists of two parts: (1) A study of Traube's area and of its extensions in front; and (2) a study in the back of the normal gastric nucleus, and of any abnormal gastric resonance combined with, or separate from it.

We may now sum up under the following headings the types of upward distension and dilatation to which reference has been made:—

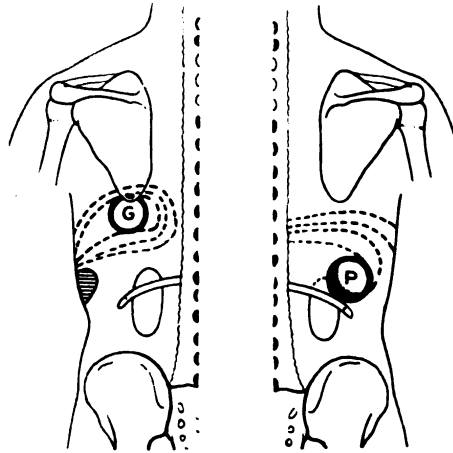
- (1) The anterior dilatation.
- (2) The dorsal dilatation.
- (3) The general or combined anterior and dorsal dilatation.

The third of these is not necessarily the most serious for the patient. The dorsal dilatation which raises the posterior wing of the diaphragm is apt to be by far the most trying to the heart.

The Technique of the Dorsal Examination for Gastric Encroachment.—It may be well to repeat that any *anterior* extension of gastric resonance works round towards the axilla, and that it may ultimately tend to work into the back. This is perhaps the most common type. Our *anterior* percussion is first to be applied over the normal Traube's area. It is then to be continued outwards and backwards until the gastric resonance comes to a stop.

A similar method is to be adopted in our *dorsal* percussion. Starting from the normal gastric nucleus, we may find that the resonance extends almost to the spine. In that case it will probably be found that it also extends towards the axilla; and in that direction there is no limit to its possible extensions.

In the familiar combined type the posterior resonance will fuse in the axilla with the anterior resonance. The gastric upward dilatation will then be traceable as one continuous resonance circling round the left half of the thorax. The diagram constructed to illustrate this progressive encroachment of resonance shows that it has an exact parallel, at the right thoracic base, in the progressive extensions of the dullness of Piorry's nucleus due to uniform enlargements of the liver.



FIGS. 12 AND 12A.

The dorsal and axillary extensions of the normal hepatic dullness in progressive hepatic enlargement, the deformations of the gastric nucleus, and the dorsal and axillary extensions of the resonance of the stomach in upward and backward gastric dilatation.

The practical conclusions arising out of the paper may be briefly summed up:—

(1) Organ-plexigraphy, long restricted to the precordium and epigastrium, still calls for greater accuracy and completeness than have yet been given to it in those regions.

(2) It should, however, no longer be restricted to them alone. In Piorry's spirit, but without his exaggerations, it should be used in any situation, and whenever it may serve a useful purpose.

(3) Dorsal plexigraphy, although too long neglected by clinicians in spite of Piorry's writings and of more recent work, affords *the larger field for normal organography*, particularly in the abdomen, as from the front, apart from the lungs, percussion is still taught to be limited to an incomplete delineation of four organs only—the heart, the liver, the stomach and the spleen.

(4) The list of its normal findings in the back includes the heart and its left auricle, the liver, the spleen, an indication of the mediastinal structures, the kidneys, the pancreas, and an indication of other mesenteric structures, besides the pelvic district. Moreover, it possesses a separate field and a method both exclusively its own in our "vertebral" or spinous-process percussion.

(5) The total range of the dorsal examination consequently exceeds anything to be obtained from the front. In reality it includes the entire contents of the trunk.

(6) For *clinical diagnosis* it is therefore not surprising that considerably more pathology is revealed by percussion in the back than in front.

(7) In addition to medical uses, dorsal percussion opens up a large field for *surgical diagnosis*.

(8) As regards "spinal" percussion and "vertebral" percussion, the surgical aspect is dealt with both in the papers of 1899 and in the paper communicated to the British Medical Association's annual meeting in 1910. This spinal aspect is far from limited, as Piorry limited it, to mere scoliosis and kyphosis. It includes something of greater importance in the shape of an earlier diagnosis of vertebral and intervertebral disease, of rheumatoid ankylosis and deformity as distinct from merely functional rigidity, of spinal neoplasms, &c.

(9) New fields for diagnosis by the surgeon and by the physician alike are opened up by dorsal percussion in three most important regions inaccessible from the front—namely, *the posterior mediastinum*, with its glandular, inflammatory, aneurysmal, tracheal, œsophageal and other affections; the *mesenteric* region, no less vital and no less remote, in connexion with the varied affections of the central abdominal organs; and the *iliac* region, which is of special importance in these days of *epidemic appendicitis*.

(10) An accurate knowledge of the normal percussion value of individual vertebræ, on the skeletal pleximetric principle, may often afford the means of detecting at an earlier date some of these centrally situated abnormalities—e.g., vertebral or glandular disease before the

formation of paravertebral abscess, œsophageal or tracheal disease before the advent of perforation and of pulmonary gangrene, &c.

(11) The value of *Grocco's triangular dullness* for the diagnosis of empyema *versus* serous effusion, of supra-diaphragmatic *versus* infra-diaphragmatic fluid collections, &c., is still imperfectly understood, but is of growing practical importance.

(12) In the field of *stomach diagnosis* a fresh departure is contained in the demonstration that normally the stomach is distantly accessible to our examination in the back. The percussion of the *gastric nucleus* will probably be regarded in the future as part of the routine of clinical examination.

(13) The existence, still practically unrecognized, of mainly *dorsal* varieties of gastric distension should render the dorsal examination indispensable in all "gastric" cases, as detailed in the paper.

(14) In conclusion, the evidence adduced (which also includes a series of original tracings taken from the chest on transparent paper, showing the normal nucleus and its enlargements and deformations, and also its belt-like fusion with an enlarged Traube's semicircular resonance) may perhaps justify this plea for a systematic teaching of "dorsal" percussion and of "vertebral" percussion, and also the opinion that this will not be successfully accomplished until the prevailing prejudice against our only instrument of precision in percussion, a suitable pleximeter, is laid aside.

DISCUSSION.

The PRESIDENT (Dr. Mitchell Bruce) said he was sure that all present had in their minds but one feeling with regard to the paper and demonstration which Dr. Ewart had given—a feeling of admiration for the immense amount of time and labour which he had devoted, not on this occasion only but for years past, to the study of percussion. He had no doubt there was much to be said by way of criticism, both of the methods and the application of the methods, but time did not remain for such a discussion that evening as the communication deserved. The one remark he would make was that Dr. Ewart's ear was perhaps a little more sensitive and accurate than the ears of some others. It required not only perfect percussion, but also very acute hearing to determine what must be, after all, very delicate shades of difference between the degrees of dullness or of resonance in the different parts of the back. He would say that the remark applied especially to the gastric nucleus behind.

They must all have percussed the back of the chest thousands of times, and yet few had been struck by the fact that there was a gastric nucleus near the left scapular apex.

Dr. GOSSAGE said he had not used pleximeters, but he wished to ask one question concerning the diagram of pleural effusion with double Grocco's triangle, where the triangles were marked out most accurately. He would like to know how it was possible to mark out the lower parts in the presence of absolute dullness due to the fluid on both sides.

Dr. EWART, in reply, said that though we might think it impossible to mark out the triangle accurately, yet on trial it would be found without any difficulty. It should be remembered that the dullness over the effusion is not a pure dullness of fluid, but a resultant of various mixed sounds conveyed by costal pleximetric conduction. For this reason the actual dullness over pleuritic effusion was not, strictly speaking, absolute. It would be more nearly absolute but for the pleximetric influence of those ribs which are still in partial contact with the lung. This mitigates the dullness. Another more telling factor was the resonance of the compressed lung, which was held anchored by the ligamentum latum pulmonis to the bottom of the spinal groove. As to the main question of plexigraphy, it should be borne in mind that in all percussions the great object was to *differentiate*. It mattered less whether an area was more dull than the adjoining one than whether its percussion was distinctly different from that of the thing from which one wanted to separate it. It might be difficult for anyone to say: "This is a flatter sound, and that is a higher note," but anyone could say, without going further, that the two sounds were judged by the ear to be quite different; and that difference was good enough for the purpose of delimitation. In short, the reciprocal triangles "of dullness" in double pleural effusion might both be in reality "shades of resonance" showing up on a background of much deeper dullness.

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COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1909-10

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Neurological Section.

November 25, 1909.

Professor C. S. SHERRINGTON, F.R.S., President of the Section,
in the Chair.

Special Sense Discharges from Organic Disease.

THE HUGHLINGS JACKSON LECTURE.

By Sir WILLIAM R. GOWERS, M.D., F.R.S.

To deliver the Hughlings Jackson Lecture is a task equally pleasant and difficult. Its pleasure and its difficulty arise from the same source. The object of the lecture is to honour the distinguished worker who is still with us, by an attempt to promote the researches which have thrown such bright lustre on his name, and, if it be possible, to carry a step further the results he has reached, or at least to provide the means that may enable others to achieve this result. But to make such an effort is to realize its difficulty. Knowledge grows most slowly, for the essential facts are rare, and those already gathered have been made to yield their lessons by the Master himself, beyond whom we cannot hope to go. I fear, therefore, my attempt will involve disappointment to you and to myself.

The subject to which I propose to limit my consideration is the symptoms of epilepsy which are related to the so-called special senses, those of smell, vision, hearing, taste, and perhaps touch; and especially the indications of such epileptic symptoms as are occasionally presented by organic disease. Examples of such epileptic discharges from organic disease, demonstrable in nature and seat, are not common. It is important to lose no opportunity of examining carefully such as are met with.

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OLFACTORY.

Anatomically, the special senses begin with that of smell, and the olfactory discharges were the first to receive special study from Dr. Hughlings Jackson, whose analytical observation has exerted a fascinating and inspiring influence on workers in every country. He published a paper calling attention to their complex and instructive peculiarities as long ago as 1866, a paper that was referred to and quoted by Sander, of Berlin, in an account of a fatal case published in 1874, when he also considered the few cases he had been able to find recorded by previous writers. Dr. Hughlings Jackson returned to the subject again and again in penetrating discussions of individual cases, especially in 1899 [3], in a paper in which Dr. Purves Stewart described six fatal cases that had been published, by Jackson and Beevor, Jackson and Colman, Anderson, Nettleship, Sander, and McLane Hamilton.

Another case was published by C. K. Mills in 1908 [6], who also added two others described by Linde [4] and by Southard [7], raising the number to nine. Two cases had been published by Dr. Buzzard in 1906 [1]; although in one of these there was no post mortem, the nature and seat of the disease were ascertained by surgery, and it is therefore deserving of inclusion. I have two other fatal cases to describe to you to-night, and I find that Sander, in addition to his own case, quoted two others, one by Lockemann [5] in 1861, and another by Westphal [10] in 1863.

The case described by Dr. Buzzard was a girl, aged 21, who was found to have a tumour in and about the right hippocampal gyrus, which had destroyed the uncus and had filled the descending cornu of the lateral ventricle. It had given rise to a prolonged sensation of a bad taste in the mouth and of a sound of bells in the head, but neither associated with her epileptic attacks. Dr. Buzzard also mentioned that the case, in which the lesion was revealed by surgery, had at first a frequent subjective sound like the loud clang of a piano, accompanied, after a time, by a very unpleasant smell and a keen feeling of reminiscence. A glioma was found infiltrating the base of the right temporal lobe, but the operation was not long survived, and no post-mortem examination was allowed.

Lockemann's case was that of a woman, aged 55, who died from a malignant tumour of the brain, which extended from the left frontal lobe to the olfactory region, and caused, for a time, peculiar olfactory warnings described as variable and not unpleasant. That of Westphal

was a syphilitic nodule in the pia mater beneath the frontal lobe, which had destroyed the right olfactory lobule and caused a very offensive smell as the warning of epileptic fits.

The case I have specially to describe is that of a man, aged 37, who was admitted to the National Hospital early in 1905, having suffered for three and a half years from frequent slight attacks, of which several occurred daily, often four or five. In these there was first a smell and taste of gas; he could not compare it to any known odour, but it seemed to be something like sulphuretted hydrogen. It caused salivation, and eructation from the stomach. The flow of saliva was so abundant that it would run from the mouth, and often filled a tea-cup. After the sense of smell he had a slight sense of turning, and if he walked he had a tendency to walk fast, almost to run. Each attack lasted from one to three minutes. Consciousness was not lost. He occasionally had a more prolonged sense of the same odour. He was a well-developed man, and his mental state presented no abnormality. His smell was defective, especially on the right side, but did not seem to be lost. There was almost complete left hemianopia. The right half field was restricted in the upper periphery, especially in the right eye. The left optic disc presented papillitis about four disk-breadths in width, but with only slight swelling (2 diopeters). The right appeared normal. The only morbid appearance it presented was a little capillary redness in the upper part. Hearing was good on each side. The tongue and throat presented no symptoms. The strength of the left hand seemed a little diminished. Both knee-jerks were brisk, but there was no foot clonus. The left abdominal reflex was slighter than the right. He had only occasional headache, not severe. There was marked tenderness to firm pressure on the right side at the temporal and lower part of the parietal region. No sound could be heard on auscultation there.

In February he was operated on by Sir Victor Horsley. The right parietal and temporal regions were exposed. The brain was bulging, but no tumour could be seen on the surface or on raising the temporal lobe, but this was firmly adherent to the dura mater near the tip. An oblique incision into the substance of the lobe revealed an infiltrating growth near the anterior extremity, which could only be partially removed, in pieces. Hæmorrhage occurred from many vessels, which had to be tied. The operation was borne badly, and the patient died a few hours after its completion.

At the necropsy it was impossible to discern the precise size and position of the growth in the temporal lobe. It was found to have

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compressed the chiasma and right optic tract; it was continuous with a mass of similar growth in the white substance of the frontal lobe. The growth was a glioma.

I shall have presently to describe other cases in which the fits were preceded by an auditory warning at first, and subsequently by an olfactory aura, consequent on the descent of a growth towards the apex of the temporal lobe.

Twelve of the cases were so extensive as to have no precise localizing value. Of these, eleven were of tumours and one of meningitis; another, that of Westphal, had apparently produced the symptom by destruction of the olfactory bulb and tract. There was extensive meningitis in the case of Southard, but there was also an aneurysm, which had destroyed the uncus. In only one case was there an isolated lesion of this structure, a small spot of softening within the uncus, and in this case, described by Hughlings Jackson and Colman, there were no sensations of smell, only the movement of the mouth and jaws, which is so suggestive of a sensation of taste. It was present also in the case of Southard, in which the uncus was destroyed by an aneurysm. Although the symptom is probably a distant secondary effect, and is present in many cases in which there is no evidence of disease in this region, we are almost compelled to regard it as evidence of a gustatory sensation. We must remember, however, that Dr. Jackson, in proposing the name "uncinate epilepsy" for these attacks with an olfactory or gustatory aura, expressly disclaimed suggesting more than the general region, traversed by the olfactory roots, as the seat of disease. Where these fibres end, in the various structures they penetrate, remains to be discovered. A putrid odour was occasionally described. Regarding this, caution is necessary when it does not attend other distinct epileptic symptoms. It may be an isolated manifestation of suppuration in one of the nasal sinuses.

Several features frequently attend these cases of epileptic attacks with an aura of smell, but little can be added to the instructive discussion they have received from Dr. Hughlings Jackson. I may mention that he has called attention to the observations of Mr. W. G. Spencer [8] on the arrest of respiration at the end of inspiration, produced by stimulation of the anterior perforated spot, over which passes the outer root of the olfactory tract and into which some of its fibres pass, as explaining the sense of dyspnoea occasionally associated with an olfactory aura. We are reminded of it by some cases of apparently idiopathic epilepsy. For instance, in one a sense of

dyspnœa seemed to have a nasal origin. The first thing was a stifling sensation in the nose, as if the patient could hardly breathe, followed by a sudden sense of a peculiar momentary bad smell.

The psychical condition which often attends the slighter attacks in cases in which there is an olfactory sensation has been termed the "dreamy" state. There seems to be only a partial impairment of consciousness, or the loss of this occurs so slowly that a distinct recollection is retained of the transient sensations which precede it. The term "dreamy" is often spontaneously applied to the memory of other slight attacks, especially to those in which some definite sensations are experienced. So also with the impression of recurrence, or of precurrence, it might be more accurately termed: the clear conviction that the same feeling has been experienced before. This is a phenomenon of normal brain states, or presumably normal, as Oliver Wendell Holmes has so well impressed upon us in his "Autocrat of the Breakfast Table," in connexion with Day and Martin's blacking. Much more strange, and at present beyond our power of conceiving an explanation, is the sense of dread, intense and causeless, which has been marked in several cases.

In ordinary epilepsy we sometimes meet with a slighter sensation of smell associated with other deliberate sensory perceptions. In one case the sound of bells, with red and blue lights, was followed by a distinct putrid odour. Another patient always had a peculiarly complex aura, a sensation in the left hypochondriac region seemed to ascend the left side of the thorax in a jerky motion, and, at the clavicle, it became a sound and could be heard as well as felt; when it reached the level of the ear it was like the hissing of a railway engine. Then he suddenly saw an old woman in a brown dress, who offered him something that had the smell of Tonquin beans. The woman then disappeared and two round lights appeared before him, which got nearer and nearer with a jerky motion, and he lost consciousness. The warning was always the same, in every detail. Such cases are, doubtless, due to a secondary involvement of an olfactory centre in a discharge beginning elsewhere, and spreading widely but slowly.

AUDITORY.

Organic disease sometimes causes attacks in which the warning is a subjective sound. As might be expected, the cause is generally a tumour of the upper part of the temporal lobe, often subcortical, beneath

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the first temporal, in which experiment indicates the situation of the auditory centre. But it will be remembered that extirpation of this has never been followed by permanent loss of hearing, neither has this result usually followed enduring destruction of this region by disease in man.

The effect of a tumour in the region was shown by the case recorded by Dr. Buzzard, which I have already mentioned. It is noteworthy that the discharge of the centre is often due to a growth in the white fibres beneath. It was so in a remarkable case which came under my notice many years ago at the National Hospital, where the patient was under the care of Dr. Hughlings Jackson, who, with characteristic kindness, has placed the notes at my disposal this evening. The patient was a man, aged 39, admitted in November, 1875. He was a painter, but without sign of lead poisoning. There was no history of syphilis. He was left-handed, but had been taught to write with the right hand. For two years he had suffered from fits, about once a week. He had also suffered much from occipital pain, increased by a cough; there was no paralysis. For the first six months there was no warning, subsequently there was an auditory aura. The slighter attacks began by a humming sound in the left ear; those of greater severity by a loud sound like a peal of bells, also to the left. The duration of the sound was about three minutes, and it ceased before the fit began, and just before it ended the patient felt a burning sensation in the left external auditory meatus and concha, like the contact of hot coal. The burning sensation then passed down the left side of the neck, and round the back of the neck to the right ear, and also across the forehead. The burning then passed down the thorax front and back, to the leg and foot, where it became a numbness. After the leg, the sensation passed down the arm to the hand. From the onset there was intense frontal pain. The head shook rapidly from side to side, and the eyelids were quivering; sparks also appeared before the eyes, always yellow. At the onset of the burning heat the face became drawn towards the left by tonic spasm, and afterwards there was trembling of the limbs of both sides. In other attacks of the same character the arm was affected by the sensation before the leg, and his description was that the arm seemed as if lying in a bed of stinging nettles. The attacks were always followed by intense headache. When admitted the optic disks were normal, but in three months optic neuritis slowly developed. Soon afterwards left hemianopia came on, the loss almost reaching to the medial line. The fits he had in the hospital were less frequent and generally slight. Each

was followed by transient weakness of the left side, the only paralytic symptom. His headache improved. After six months he went out and returned at the end of October, 1876. The fits had continued, many had occurred without warning; but those which he had after re-admission presented the same aura, the sound of a peal of bells, now followed by pain at the back of the left hand, which passed up the arm to a little above the elbow, and was then felt in the left side of the face. Tingling followed in the left lower leg, and passed up the thigh; then he fell unconscious. The arm was the seat of constant tremor, which was present also in the leg when he was sitting, but ceased when he stood. It began suddenly in June in a violent form soon after rising. In the arm it was coarser than in paralysis agitans, and involved all the joints, but was greater in the wrist and fingers than in the elbow or shoulder. A movement stilled it for a time but it soon returned. The left arm had become distinctly weak; the grasp was 35 with the right hand, and only 10 with the left; the leg seemed strong. The optic neuritis was subsiding, and vision was considerably impaired.

He did not then stay long in the hospital, and the notes are brief, but he was again admitted in October, 1878. Continuous occipital headache still troubled him. Left hemiplegia had gradually developed and had attained a considerable degree. The neuritis had led to consecutive atrophy with still more impairment of sight, greater in the right eye. The left hemianopia persisted. Hearing was noted to be good on each side; smell also was good and equal; taste seemed diminished. His fits had become less frequent and many appeared to have been bilateral. He still had frequent severe occipital headache. Many fits occurred after he returned to the hospital, the description of them in the notes never now mentioned an auditory aura. In those that were severe the convulsion was bilateral, but more severe in the left side.

In the beginning of 1879 sight had almost completely failed. Movement of the eyes to the left was attended by nystagmus. Hearing was impaired on the left side, the watch not being heard at all, while it was clearly heard on the right. The left arm was quite powerless; the leg could still be moved, although feebly, but it was not rigid. The right leg could be moved freely. Sensation was not much impaired in the left limbs. The occipital headache was slighter, but it afterwards again became severe. During the next few months he had an occasional slight fit, and then continuous delirium set in. His wife was dead, but he asserted that he could see her practising prostitution with other

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patients in the ward. He was quite blind, but when assured that it was not so, his reply was, "Do you think I cannot believe my own eyes?" In August there was alternate convulsion in the right and left side of the face, and then a severe convulsion on the right side. In September the delirium ceased. Smell was noted to be still deficient, but "better on the left side than on the right." Occasionally, however, he had an unpleasant subjective smell "like a strong liniment, almost taking his breath away." On the right side hearing was still good, and on the left the watch could be just detected in contact; there was often subjective ringing. Taste was slow; occasionally a subjective bitter taste was experienced. Sight was practically gone; occasionally he still had subjective sparks to the left. The chest moved well on the right side, very little on the left. He spoke at first in an audible tone, but his voice soon died down to a whisper. The left arm was powerless, with no rigidity, but he complained of a dragging pain in it, and if moved away from his side he could not tell where it was. The right arm was moved freely, but was too feeble to act on a dynamometer. The left leg was powerless but without rigidity, and passive movement caused much pain in it. The right could be well moved. The right knee-jerk was present, the left very slight, and the attempt to obtain it caused pain. There was no foot clonus. He continued in this condition, having a few fits, generally slight. In one, the convulsion was in the right arm and slightly in both legs. On Dec. 7 he died suddenly.

Post Mortem.—A tumour was found in the right hemisphere, but no morbid process in the left, or in the pons. The growth lay between the optic thalamus and corpus striatum on the inner side, and the convolutions of the temporal lobe on the outer. It was almost confined to the white substance, ceasing about a twelfth of an inch from the grey substance of the temporal convolutions and of the island of Reil, but it had invaded the outer segment of the lenticular nucleus behind the middle of the corpus striatum, and compressed the other segments. It had also invaded the posterior extremity of the optic thalamus, for $\frac{1}{8}$ in. only, and the thalamic substance was softened in an area of $\frac{1}{4}$ in., just behind the anterior extremity. A tongue of growth appeared at the base between the crus and the uncinate gyrus, which was displaced outwards but not invaded superficially. It had compressed but not invaded the optic tract almost up to the commissure. Behind, in front of the corpora quadrigemina, the growth had invaded the crus in an area the size of a split pea and thence had passed into the anterior, and slightly into the posterior, right quadrigeminal bodies, but had not crossed the middle

line. The anterior limit of the growth was the neighbourhood of the grey matter of the convolutions of the island of Reil. The highest level it reached was a little above the floor of the lateral ventricle, outside it. It had filled the descending cornu, and extended beneath the anterior half of the posterior cornu, where it ceased by a rounded end, which was separated by a thin layer of softening from the cerebral substance. Nowhere else was there this separation. The growth was, for the most part, similar to the appearance and consistence of the grey matter of the cortex. An exception was presented by the region between the posterior part of the optic thalamus and the temporal cortex; here it was much firmer, and almost cartilaginous in consistence, yellowish in tint, and it contained several small irregular cavities. It was evidently the oldest part of the growth. The structure of the tumour presented the aspect of a glio-sarcoma. It was composed of small oval cells, with a few fibres.

The features of this case are of great interest. The earlier fits must have been due to discharges in the right hemisphere, beginning with the auditory centre, as the result of the growth in the white fibres beneath it. Although the cortical centre would be isolated, the discharge would affect consciousness as a sound, and extending to the motor region, which was not isolated, would cause convulsion on the left side. But before the convulsion, the discharge seems to have spread to the centre for common sensibility in the ear, the external auditory meatus and concha, and thence to that for the neck, and the trunk and the left limbs. The discharge in the centre for common sensation in a special sense organ, as well as the special sense itself, is rare, but is occasionally met with in epilepsy. I have not otherwise met with it as a consequence of organic disease. It is, perhaps, intelligible in relation to the auditory centre, because the vibrations of touch stand in so close a relation to those of sound. Lastly, a feature which is difficult to explain is the final convulsions on the right side, for which no explanation could be found in any lesion in the left hemisphere. We must consider that they were produced by the tumour in the right hemisphere acting on the left cortex through the fibres of the corpus callosum.

Dr. Colman [2], in a very instructive paper on hallucinations produced by organic disease, has published an account of a case of a tumour of the lower part of the central convolutions and the adjacent first temporal on the left side, in which there was the sound of bells in the right ear and afterwards a pleasing hallucination that a musical box was constantly playing. Dr. Colman has also referred to the case of

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a Jewess, of Odessa, who was subjected by her husband to blows on the head, which amounted to a rough physiological experiment, the results of which were described by Tomaszewski and Ssimonowitsch in 1889 [9]. Congestion of the cranial bone, thickening of the dura mater and pia mater, and inflammatory tissue beneath the latter, with changes in the cortex, were found over part of the right central convolution, the posterior half of the first temporal, the supra-marginal and the angular gyrus. She suffered from convulsions, chiefly left-sided, and persistent auditory sensation on the left side, as well as left-sided visual hallucinations.

Last year, an Italian widow, aged 39, was under my care at the hospital with symptoms of a tumour of the temporal lobe, double optic neuritis, headache, and convulsions, some preceded by the sound of a bell ringing on the left, by a very offensive smell, and the vision of a strange woman. Her case was especially fixed in our memory by the fact that she was quite willing that her head should be opened, but she had a profound objection, at first insuperable, to the removal of her hair; she consented only on the assurance that it need only be taken off on one side, and that it would surely grow again. Sir Victor Horsley operated in July. A round encapsuled tumour, subcortical, was found in the temporal lobe; it reached down almost to the uncus. It was shelled out, through an incision, with as little injury to the brain as possible. She had afterwards slight left hemiplegia and diminution of sensation, but she left after two months, rapidly becoming well. Her only subsequent fits were on the day of the operation, and in these the previous aura, the offensive smell, and vision of a strange woman, occurred after the cessation of ten minutes' spasm of the left arm, not before the motor convulsion. These warnings had occurred only for a year and a half, when the fits had been more frequent, but the first fit was when aged 14—a month after her marriage—and they had occurred two or three times a year ever since, with tongue-biting and micturition, quite similar to those of the last year. No family tendency could be heard of. When aged 19 she had a small tumour removed from the breast. The cerebral tumour was thought to be a glioma; its structure suggested a long duration, and it may well have been present at the time of the first fit and have caused the convulsions, which were only associated with the auditory and olfactory warnings after it had attained a considerable size.

The reverse is the case with a woman who has just undergone the first stage of operation (on Tuesday last). Her age is 35. Fits have occurred for seven years, and were associated long ago with a smell of

rotten fish and also the sound of a voice, but these have been absent now for several years. Their cause may be learned next week.¹

VISUAL.

Discharges in the visual centre seldom result from organic disease; the half-vision centre in the occipital lobe is still the only one generally recognized. Many years ago I suggested that the symptoms of disease give weight to the experimental conclusions of Ferrier, which indicated that the region of the angular convolution between the parietal and occipital lobes has a visual function. The facts suggest, I think, that this region is connected with both half-vision centres in a complex way, so as to represent the visual function of both eyes, but of the opposite eye to a greater extent than of the eye of the same side. Partial disease of this centre seems to lower its function as a whole. In the hemi-anæsthesia of hysteria this centre seems to be put out of action, and the effect on vision is well known. Such an hysterical condition is commonly regarded as not worthy of consideration, but functional states are the only evidence we can obtain of some arrangements in the central nervous system; a similar condition of vision may result from an organic lesion.

Some of the preliminary spectra of migraine can be best understood on the assumption that they depend on a slow discharge in this higher visual centre, on the side in which the subsequent headache is felt, especially those that have the form of what is termed the "angled fortification spectrum." The inhibition which accompanies the spectra seems sometimes to be seated in this centre, sometimes in both (when there is a central loss), sometimes in the related half-vision centre. But it is remarkable how very seldom inhibition of the latter forms part of the warning of idiopathic epilepsy.

I must not allow myself to be led into a discussion of functional affections, but I have to describe some cases of organic disease involving the angular gyrus and causing visual symptoms—cases which seem to afford definite support to the opinion I have put forward. The first case is that of a man, J. S., aged 45, who is left-handed and thinks he became so, except for writing, in consequence of an accident to the right hand at the age of about 10. He has served in the Navy for twenty years, and during that time he had many blows on the head.

¹ It turned out to be a very large cyst in the temporal lobe, connected with the lateral ventricle.

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He denied syphilis, but admitted many risks. Some time in 1902 he was struck on the head by a heavy piece of coal, and in November of that year—how long after the blow he does not know—he had a severe fit, which compelled him to stay in bed five days. He remembered, during this time, seeing a bright, white light on his left. Subsequently he had slight attacks in which his head turned to the left and there was brief loss of consciousness. In March, 1904, he had three severe fits in one day, with convulsion on the left side and coloured lights to the left. Afterwards slighter fits occurred every few days until his admission into the hospital in May, and afterwards.

He was a man of considerable intelligence. His smell, taste, sight and hearing were normal, and so were the optic disks and ocular movements. The left arm was slightly weak; the grasp 75 compared with 90 on the right side, although in general left-handed. The left knee-jerk was the more active, and the plantar reflex was extensor, the right being flexor. The attacks occurred frequently, but varied much in degree. Very careful notes were taken by Dr. Gordon Holmes, who was then resident medical officer. In the slightest, the eyes became fixed without deviation. The left side of the face was the seat of slight tonic spasm, and the head turned slowly to the left; consciousness seemed dull for about a minute. In others, in addition, the left arm became rigid, the elbow a little flexed without movement of the wrist or fingers. Smacking movements of the lips terminated each attack, but no sensation of smell could be ascertained, although sometimes he spoke of an unpleasant bitter taste in the mouth.

Many attacks, especially those that were more severe, were heralded by the appearance of several small round objects, seven or ten, just to the left of the fixing point and above it. They were brightly coloured, red on the outside; within this was a zone of blue, the centre being white. They were always the same, and they moved to the left or returned in a series of jerky movements for five or seven minutes, often longer, after being first seen. During this time his eyes were constantly being jerked towards the left. Almost immediately after the appearance of the balls, dimness developed in the left half of the field, which was slight at first, but rapidly increased in degree, until it amounted to complete hemianopia, extending to the middle line, except in a small area round the fixing point. It remained complete until about five minutes after the coloured balls disappeared, when it slowly lessened. At first, soon after the disappearance of the balls, round markings the size of the balls could be dimly seen at the place these had occupied.

The passing away of the hemianopia was slow; a little dimness still remained half an hour afterwards.

In some attacks there was initial jerking of the eyes to the extreme left, and of the head, by tonic spasm. After a few seconds the spasm ceased, but presently came on again, and these recurrences went on for twenty minutes, each being attended by the noise in the mouth as if the patient were chewing; the arm and leg were rigid, and the arm was the seat of a fine tremor. In it there was complete loss of sensation, to touch and pain, up to 2 in. above the elbow-joint. The right limbs were normal. During this time the patient saw the coloured balls to the left for about ten minutes. When they disappeared the eyes and head could be moved to the right. The tremor of the arm ceased, but was replaced by irregular motion, comparable to athetosis. In a few minutes the loss of sensation had diminished, and extended only over the hand up to 2 in. above the wrist. Consciousness was unimpaired, but after an attack he frequently used wrong words in speaking, which bore no resemblance to those he intended to say. Such an attack was sometimes followed by a recurrence, and in this he sometimes completely lost his sight for about a minute, when the half vision returned. Occasionally he felt only the loss of sensation in the arm, which was verified by examination, and there was extreme deviation of the eyes to the left, without the appearance of the coloured balls. The fields were many times separately taken with the perimeter and always found normal.

The following note was made of his impairment in speech: "He always understands the nature of objects and can easily express all his ideas and wants, speaking quite correctly. He understands everything said to him, even when spoken to quickly or in complicated sentences. He can read fairly well, but says it is much more difficult than it was before he became liable to fits; the effort tires him. He can see the words quite well, but has to read a sentence over and over again before he can understand the meaning. His right hand being good, he can write quite well and legibly, but complains of difficulty in expressing his thoughts in writing."

In July he was operated on by Sir Victor Horsley. The dura mater over the supramarginal gyrus was found on incision to be considerably thickened; it was removed. No tumour could be seen on the surface, but the pia mater was milky, opaque and thickened, and the surface of the brain was firm and nodular, and hard to the touch. A horizontal incision was made just above the supramarginal convolution, and the substance of the brain was evidently sclerosed, greyish white and opaque in aspect;

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but, though the incision was deep enough to open the lateral ventricle, no tumour was found. In the angular gyrus, posterior to the incision, a firmer patch of sclerosis was found. The operation was well borne.

One or two fits occurred each subsequent day on the left side, beginning in the face with clonic spasm. Consciousness was not lost. After a week they became less frequent. His mental state was very obtuse. During August there was steady improvement. The wound healed firmly and his fits became less frequent, and ceased by the middle of the month. In the middle of September it was noted that his mental state was good and bright, but now and then he seemed to become dull and strange, as though he had a slight attack of petit mal. Hearing was good, and the only defect in sight was a slight peripheral restriction in the left lower edge of the field. The two knee-jerks were equal and the plantars both flexor. Spontaneous speech was quite good. He still complained of difficulty in reading and that sometimes he could not understand what he read, but he read short sentences quite well and showed that he understood them. In reading aloud he made many mistakes, especially with the longer words, and even short ones were often pronounced wrongly. Spontaneous writing was with many mistakes, which he recognized on reading it afterwards. Writing from dictation was very imperfect and long words he said he could not write, and when urged to try he was unable to produce anything like the correct word. He seemed, indeed, to produce some retained visual symbols rather than to translate the auditory symbol into one of appropriate visual character. By the middle of October all trace of hemiplegia had disappeared. He could write intelligently to his wife, but still failed in writing from dictation. He was discharged October 18, 1904, and has since been heard of as able to pursue his work in Ireland.

The visual phenomena present in the attacks of this patient seem best explained by a discharge of the assumed higher visual centre in the region of the right angular gyrus, a discharge which spread to the adjacent motor centres, or to it from them, and also produced temporary inhibition of the right half-vision centre, which very slowly passed away after the cessation of the discharge. They show how readily the half-vision centre undergoes inhibition, and that sometimes a secondary discharge of the centre in the other hemisphere may result. This has evidently occurred when a hæmorrhage in one occipital lobe has caused permanent hemianopia, but at first complete loss of sight resulted by the inhibition of the half-vision centre in the other hemisphere, which, in one case I have seen, continued for a week.

Another case has been in the hospital, in which a traumatic superficial lesion of the angular gyrus gave rise to fits beginning with a visual aura. The patient was under the care of Dr. Batten, to whom I am greatly obliged for the opportunity of describing it to you. She is a girl, now aged 9, without neurotic heredity, who when aged $3\frac{1}{2}$ fell down fourteen or fifteen stairs and struck her head on the stone tiles of the floor below. There was no cut or bruise of the head, and the exact place of the blow was at the time uncertain. She seemed to have no loss of consciousness. Half an hour later she had a fit, with loss of consciousness and twitching of the right side, face, arm and leg, and she vomited several times during the convulsion. When it ceased she went to sleep, and after two hours woke up seemingly well, and no loss of power was noticed. Similar attacks recurred, and during the following year she had about forty. Then they ceased and she was free for three years, until Christmas 1907, when they returned, and have frequently recurred since in severe form until she came to the National Hospital on June 15, 1909. In the hospital she had several attacks differing in severity, but similar to those which had occurred previously. Each began with a bright light in the right of the visual field, and she always rubs the eye with her right hand, not, she says, on account of any discomfort in the eye, but to get rid of the light; the head is then turned slowly to the right and the eyes also deviate to the right. Consciousness is then lost. Tonic spasm then occurs in the right side, followed by clonic movement and twitching of the right side of the face and jerking of the eyes. The duration of the whole is two minutes. Consciousness then returns. Immediately afterwards there was loss in the right part of the field of vision, quickly passing away, and diminution of tactile sensibility in the right hand and extensor plantar reflex.

Immediately after the fit, sight was distinctly lost in the right side of the field; it was soon regained, although for a time she said she saw objects more distinctly in the left than in the right half. Sensation in the hand was lessened to both touch and pain, and even after a quarter of an hour she complained that she could not feel the hand properly. The right plantar reflex was extensor after the fit, the left flexor, and the abdominal reflex was much less on the right side than on the left. In one severe fit there was also some spasm in the left leg. The visual spectrum, which she calls "the twinkles," always appears on the right, and she always rubs the right eye. They seem accompanied by deviation of the eyes to the right. She says the twinkles are "like stars."

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She once said to her father: "They are all colours, green and red and all, and there is a mist over my eye." She generally denies that the spectrum is coloured. Apart from the attacks, her physical condition seemed quite normal. Her ocular movements were perfect, and so, usually, were all reflex actions, including the abdominal and plantar.

On July 6 she was trephined by Mr. Sargent over the left parieto-occipital region. On exposing the brain the angular gyrus appeared paler and wider than the other convolutions, and in its posterior part was softened. On incision a dark grumous material escaped as well as some pale gelatinous substance. It seemed to be an old blood-cyst. It occupied a small region around the termination of the parallel fissure. The wound healed well. The attacks, however, returned and she has been readmitted. They begin with the same visual aura, but the convulsion is now general.

I am indebted to Sir Victor Horsley for the facts of another case. An officer received a severe blow on the right side of the head in the hinder parietal region, which caused fits with a visual aura to the left. He was trephined, and laceration of the surface at the region of the angular gyrus was found.

In this connexion I may refer to the case I have already mentioned of the Odessa Jewess who had a persistent visual hallucination to the left, and the traumatic meningitis on the right side of the brain had involved the whole region of the angular gyrus.

These cases all speak with the same clear significance. It is different from the diminution of the remaining half-field in some cases of hemianopia, which was the evidence on which I relied in my *Manual*, as justification of the assumption in man of the higher visual function of the region of the angular gyrus. The higher the level of function, the more complex its representation may be, and we may hesitate to reject entirely that of which the evidence may still seem imperfect.

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Neurological Section.

December 9, 1909.

Dr. THOMAS BUZZARD in the Chair.

Recurring Left-sided Convulsions, with Transient Motor Aphasia, in a Left-handed Woman.

By JAMES TAYLOR, M.D.

A. A., FEMALE, aged 36, married, admitted April 20, 1908 ; no children, one miscarriage at seventh month. No history of lues in husband ; nothing significant in family history. In April, 1908, convulsion ; fell down, face and shoulder twitched, was unconscious. Repeated convulsions of same character, did not speak for four days, then noticed to be paralysed, left arm and hand. Slow return of power. On admission, nine days after onset, slight weakness left face, arm and leg ; deep reflexes more active on left than on right side, plantars flexor ; slight motor aphasia. On day of admission seven fits, left-sided, commencing in face, and several on two following days ; gradual improvement, and went out practically well a month after admission. .

Readmitted August 20, 1908 : Headache, vomiting, unintelligible speech, and occasional spasm and tremor in left arm for ten days previously. On day before admission several *right*-sided fits. On admission no local weakness, deep reflexes equal and active, plantars both extensor. During first three days in hospital had eleven fits, all right-sided at the start, but becoming general ; gradually recovered and left hospital in October, 1908.

In September, 1909, left-sided fit in the night. Next day had a series of similar fits ; was paralysed on left side after them. Readmitted on September 29, 1909, and was found to have some weakness in conjugate movements of eyes to left ; weakness left face, and paralysis (complete) of left arm and leg. The deep reflexes were more active on left than

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on right; the left plantar was extensor, the right flexor. There was some difficulty in speaking and reading. For some days after admission many left-sided fits. Gradual improvement; no optic neuritis at any time. The patient is left-handed.

Left Hemiplegia and Left Third Nerve Paralysis.

By JAMES TAYLOR, M.D.

S. E., FEMALE, aged 34, single, admitted under Dr. Beevor on October 6, 1908, complaining of drooping left eyelid and diplopia. Mother, two brothers, and one sister healthy; father dead, cause unknown. Previous history unimportant except for amenorrhœa and sore throats during last two years. In November, 1907, sudden weakness of left face, arm and leg; gradually passed off. Eight weeks before admission, headache, drooping of left eyelid and diplopia; two or three weeks later, diplopia, vomiting—especially in early morning. On admission, complete left third nerve paralysis, weakness of left face. No change in limb reflexes. Nodes on forehead, corneal opacities suggesting congenital syphilis (Marcus Gunn); teeth normal. Discharged December 13, 1908, improved in general condition; ocular condition unaltered.

Remained fairly well till March, 1909, then giddiness and vomiting commenced. Admitted to St. Thomas's in July; remained there eleven weeks without improving; giddiness and vomiting have continued since. Readmitted September 22, 1909, under my care. On admission, third nerve paralysis still present; conditions otherwise unchanged except for headache and vomiting. Under treatment she has improved a good deal in respect of these symptoms; the ocular condition continues.

Case of Tremor.

By E. FARQUHAR BUZZARD, M.D.

T. W. C., A MAN, aged 59, first came under observation in September, 1905. He was complaining of tremors affecting his four limbs and his head, which had commenced gradually about five years before, and which had increased in intensity. The tremors resembled those of paralysis agitans, but the patient had not the facies, the posture, or gait

of that disease. He had had a primary sore at the age of 20, and had been well until a few months before the onset of the tremors, when he had a severe illness, in the course of which he had developed a right facial palsy of peripheral type, a transient diplopia, and severe abdominal pain. There was no evidence that he had been exposed to lead poisoning.

On examination he was found to have Argyll-Robertson pupils, the remains of the facial palsy, but no other affection of the cranial nerves. He showed no paresis or rigidity; sensibility was intact; his tendon-jerks were exaggerated and his plantar reflexes flexor in type. There was no ataxy. His sphincter control was perfect.

During the four years which have elapsed since he was first seen, his tremors have diminished rather than increased, his tendon-jerks have become very difficult to obtain, but his plantar reflexes have remained flexor in type. He now walks with some difficulty on rather a wide base, and describes a sensation of his feet sinking into the ground. Romberg's sign is not present. There is no loss of power and no definite defect of cutaneous or deep sensibility. The tremors are somewhat increased by voluntary movement, although constantly present during rest.

DISCUSSION.

Dr. FARQUHAR BUZZARD said there could be little doubt that the patient was suffering from tabes. It was an interesting and difficult problem to decide whether the tremor was due to concomitant paralysis agitans or whether it was the result of some syphilitic lesion of the mid-brain, possibly of the thalamic region. The combination of tabes and paralysis agitans was very unusual, but its possibility raised the question as to the exact nature of the result produced by two conditions more or less antagonistic in their effects. Would the hypotonia of tabes exert a solvent action upon the "starchiness" of paralysis agitans? Against the diagnosis of paralysis agitans were two facts: in the first place, the tremor had existed for at least nine years without becoming intensified; in the second place, the tremor had been general from the first, and not hemiplegic in onset, as was nearly always the case in that disease.

Dr. JAMES TAYLOR said that a number of years ago he saw, in the Golden Square Hospital, a woman who attended on account of attacks of breathlessness and difficulty in breathing, for which she had had tracheotomy done. It was a definite case of tabes, with Argyll-Robertson pupils, as well as laryngeal crises and absence of knee-jerks, but without ataxy. But she was a very typical case of paralysis agitans; so that at least in that case the conjunction of two

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diseases had apparently not had any effect in dissolving the ordinary clinical picture of paralysis agitans, as she had the facies, the typical gait, and the tremor. He thought Dr. Buzzard's view of the case shown was correct, in regarding the tremor not as of the paralysis agitans type, but rather as the result of some conjoint defect in the thalamic region, perhaps associated with tabes, and probably of syphilitic origin.

Case of Compression Paraplegia: Syphilitic Meningitis.

By T. GRAINGER STEWART, M.B.

S. B., MALE, aged 44, married. Complaint: Weakness of the legs and incontinence of urine. Family history unimportant. Previous health good, save for venereal disease twenty years ago.

Present illness: Eighteen months ago patient injured his left knee and was confined to bed for some weeks. On getting up he noticed that his left leg was weak; this weakness gradually increased, and at times when the patient was walking "the left leg seemed to give under him." As the weakness progressed he noticed that at night-time the left leg "started up" of its own accord. Shortly after this the leg felt numb. Two months ago the right leg began to get weak, and at the same time he noticed that he had difficulty in retaining his urine. Since then the weakness has increased, both legs have become numb, and the sphincter trouble more severe. The patient can walk alone with the aid of a stick.

Present state: The cranial nerves are normal, except that both pupils have an impaired reaction to light. The patient has nothing wrong above the level of the eleventh dorsal segment, but from this level downwards he has a spastic paraplegia affecting the left leg more than the right. Reflexes: The deep reflexes of both lower limbs are increased, with clonus on the left side. Both plantar reflexes are extensor in type, and the superficial abdominal reflexes are absent below the level of the tenth dorsal segment. The sphincter trouble is incontinence of urine and constipation. The patient feels his water passing. Sensory: There is a relative diminution to all forms of cutaneous sensibility below the level of the tenth dorsal segment on the left side, and below the level of the eleventh dorsal segment on the right. Deep sensibility is not impaired, and the sense of position very slight, if at all. Within the last few days the patient has noticed a tight feeling round the lower abdomen. Spine normal, no tenderness on pressure.

DISCUSSION.

Dr. GRAINGER STEWART said he purposely did not call these cases meningo-myelitis, as he thought the signs and symptoms referable to the cord could be explained simply by pressure on the cord from constriction by a localized meningeal thickening. In many similar cases which he had seen operated upon the appearances of the cord and membrane were somewhat disappointing. In a certain number of cases there was a well-recognized gummatous thickening of the membranes, in others the pia arachnoid was somewhat thickened and opaque-looking, but not more than in cases in which syphilis was excluded. In others there was adherence between the pia, the arachnoid, and the dura, with damming up of cerebrospinal fluid, which was released by opening the dura and breaking down adhesions. Whether these cases should be called syphilitic meningitis with compression of the cord, or meningo-myelitis, was a point to be decided by the clinical history and by the state of the patient. In a certain number of cases there were cord symptoms resulting from a condition affecting the membranes alone, the symptoms passing off immediately after the meningeal pressure was relieved. As an example of that he could recall a case on which Sir Victor Horsley operated, that of a woman who, for four months, had had pain, first in the region of the second dorsal posterior root, and, later, extending to involve the posterior roots on the left side, from the eighth to the fourth dorsal inclusive. First, she had pain, and then loss of sensation in those root areas. When he (Dr. Stewart) saw her she had definite signs of pressure on the left side of the cord, the abdominal and epigastric reflexes were absent on the left side, and on several occasions there was an extensor response on the left side. She also had slight sensory loss over all parts below the level of the second dorsal segment. That was compatible with pressure on the cord, as immediately after the operation she recovered completely from all the cord symptoms, which seemed to show that there was no myelitic condition inside the cord. If the pressure was prolonged he thought that changes were set up inside the cord, either by strangulation or by local interference with the blood supply, resulting locally in a chronic sclerosis or thrombotic softening, subacute in onset. In his opinion, some of the cases thought to belong to the group which Erb described as due to primary degeneration of the cord were in reality cases of syphilitic meningitis with pressure on the cord. The results of operation had been so wonderful in some cases that more attention ought to be devoted to the subject. The second case was that of a man who had syphilis when very young, and who had not been thoroughly treated. Five years ago he began to have progressive weakness of both legs, first of the right. He now had spastic paraplegia and some slight motor weakness. He presented the typical reflex changes of spastic paraplegia and diminution of all forms of sensation up to the level of the fourth dorsal segment. Both pupils were "pin-point" and inactive to light. He thought the patient had a meningeal condition which had caused compression of the cord and led to changes inside the cord. He did not expect that operation would improve the patient's condition.

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Dr. F. E. BATTEN asked how Dr. Stewart proposed to distinguish between cases which he called meningeal and those which he called myelitis, especially when they occurred in the mid-dorsal region; he could understand the distinction when they occurred in the upper dorsal and cervical. But the importance of distinguishing between them was great, because Dr. Stewart said operation would benefit one class of case but not the other. He (Dr. Batten) had had three such cases under his care, and all with symptoms pointing to a gradually increasing paraplegia occurring below the mid-dorsal region of the cord. In all of them operation was performed. In one some fine adhesions were found about the cord, and the man made a good recovery, regaining power, sensation, and control over his bladder. In the second case there were no adhesions, the cord looked thin and wasted, there was much fluid escaping, and there was no improvement. In the third case the symptoms had been gradually coming on, and no improvement followed operation. So it was difficult to know how to distinguish between the cases in which there was thickening of the membranes, and those in which there was myelitis; certainly they were not of the sudden onset which Dr. Stewart suggested often occurred in myelitis cases.

Dr. HINDS HOWELL said that recently he had made an autopsy on a similar case. There was paraplegia due to a lesion in the dorsal region, and operation was performed. On opening the dura there was a gush of fluid, and pachymeningitis was described as present; nothing further was done at the operation, beyond breaking down the adhesions between the dura and the leptomeninges. The patient died of bronchitis. At the post-mortem he found comparatively little thickening of the meninges, but there had been some adhesions between the dura and pia arachnoid which had dammed up the cerebrospinal fluid, and as a result there had been compression of the cord. The microscope showed that the meningeal thickening was not a gummatous infiltration, but the result of other inflammation. The spinal cord showed no degeneration by the Weigert-Pal method and very little with Marchi's fluid, and the local condition of the cells was fairly good. That would probably have been a successful case if the patient had not died from an accidental complication. It was easily understood why certain of the cases did not do well, although there might be no accompanying myelitic condition of the cord. If the pressure had lasted a considerable time, and had been sufficient to cause degeneration in the spinal cord of a permanent type, one could not expect to get very marked improvement as the result of operation.

Dr. GRAINGER STEWART, in reply, agreed with Dr. Batten that it was very difficult to know when there was myelitis or meningitis; but he suggested that in the meningeal cases there was a gradual onset, usually unilateral, one leg being affected first, without sphincter trouble. The other leg became affected later; and commonly the sphincter trouble came on when both legs were affected. There was, at the level of the lesion, a

greater degree of sensory impairment than lower down, and that might be taken as evidence of pressure on the posterior roots, as the local sensory changes were more peripheral in type. In myelitis the case was usually bilateral, and the onset was more rapid and there was no evidence of a local meningeal condition, extending to perhaps two or three segments of the cord. As a rule, in pure syphilitic myelitis there was immediate benefit from antisiphilitic treatment, but that was not so in meningitis. The reason operation was recommended was that in regard to the nervous system one could not afford to neglect the fact that in such cases it was not the syphilis which was being dealt with, but an attempt was being made to prevent damage to the nervous system proper. He held that in those cases the damage to the cord was not primarily syphilitic, but was secondary to the pressure exerted on the cord, and every effort should therefore be made to relieve that pressure. Moreover, after operation antisiphilitic treatment was often more efficacious. He could not say why that was, but it had been observed by many.

Two Cases of Chronic Syphilitic Poliomyelitis.

By WILFRED HARRIS, M.D.

CASE I.

C. W., AGED 40, married woman, in July, 1907, noticed pain for a short time in the left upper arm, followed soon by weakness in raising the arm and in bending the elbow and in extending the fingers of the left hand. Twelve months later pain followed in the right arm, and soon weakness of the right arm similar in character to the left. Eight months ago the condition was very similar to that now present, but she could just hold up the right arm above the head, and there was then well-marked reaction of degeneration in the wasted muscles of the left arm.

Present state: Complete and symmetrical paralysis of the muscles supplied by the fifth and seventh cervical segments, viz., both deltoids, spinati, biceps, brachialis anticus, supinators, pronator radii teres, radial extensors of the wrist, and the extensors of the fingers and thumbs. She has no pains whatever now, and the sensation to all forms is normal. No headache or diplopia. No sphincter trouble. The lower limbs and the gait are quite unaffected. The knee-jerks and Achilles-jerks and the forearm-jerks are all absent. Jaw-jerk present. The pupils are sluggish in reaction to light, especially the left. Lumbar puncture shows moderate lymphocytosis only. Married seventeen years; one healthy child, aged 16. No miscarriages. Wassermann reaction positive.

Treatment by injections of strychnine, and later by mercury and iodide, have produced no improvement.

CASE II.

J. K., aged 60, bootmaker. Three and a half years ago pains in back of neck and in right shoulder, followed by wasting of the muscles of the right arm and forearm. The weakness of the right arm increased so much that after six months he could not raise the right arm or feed himself. This improved considerably, and in January, 1907, he could raise the right arm and bend the forearm fairly well. His condition then was much the same as now, except that two years ago he developed weakness of the extensors in the left forearm.

Present state: Wasting of the posterior fibres of both deltoids, and slight weakness of the right serratus magnus. Right forearm: Marked wasting of the extensors of the fingers, the radial extensors of the wrist, and of the thenar eminence. All the other muscles normal. Left forearm similar, but much less marked weakness of the extensors of the fingers, the radial extensors of the wrist, and the thenar eminence. The biceps and supinators, the extensors of the thumbs, and the ulnar extensor of the wrist on each side are quite normal. Electrical reactions: The wasted muscles scarcely respond at all, either to strong faradism or to galvanism, with the exception of the left thenar muscles, which show reaction of degeneration. The general appearance of the atrophy resembles lead neuritis, but there is no lead line and he has never worked in lead. He has no pains whatever now, and has never had diplopia or sphincter trouble. Bilateral Argyll-Robertson pupils. Knee-jerks and Achilles-jerks brisk. No trace of analgesia or anæsthesia. Lower limbs and gait normal. History of venereal discharge forty-two years ago. Wassermann reaction positive. Lumbar puncture showed no excess of leucocytes.

Chronic Meningo-myelitis: Internal Ophthalmoplegia.

By WILFRED HARRIS, M.D.

V. W. C., AGED 32; denies all venereal disease. Six years ago jarred his spine in falling off a sofa, noticed a numb feeling in the back immediately afterwards, and has never been well since. Six months later he noticed pains around the upper part of the abdomen and "sore pains"

in the legs. Three and a half years ago he noticed the vision of the left eye was impaired, and it was then found that the left pupil was dilated and fixed, and there was complete loss of accommodation in the left eye; three months later severe neuralgic pains at the back of the eyes. In September, 1906, the left pupil was dilated and fixed, with loss of accommodation, and the right eye was quite normal; nervous system in all other respects normal. Two months later the vision of the right eye also failed for reading, and both pupils were now dilated and fixed, with bilateral loss of accommodation. The pupils contracted well under eserine and the power of near vision returned temporarily; ocular movements perfect; never any diplopia; slight left voluntary ptosis. In February, 1907, the right pupil recovered partially and the power of accommodation returned in that eye. In November, 1907, developed feeling of tightness in the perinaeum and backs of thighs, and legs became weak. Entered Maida Vale Hospital, where he became worse for the first three weeks, numbness of the legs increasing, and complete retention of urine for ten days. Double ankle clonus and extensor plantar reflexes now appeared. Under inunction of mercury and biniodide internally, he then steadily improved and the legs became practically well. There has always been some slight hesitation of the bladder since.

Present state: Left internal ophthalmoplegia, pupil widely dilated and fixed; right pupil medium small, reacts to strong light, and accommodation normal; knee-jerks and arm-jerks brisk; right Achilles-jerk brisk, but the left is diminished; diffuse analgesia on legs, arms, and trunk; Wassermann reaction positive.

DISCUSSION.

Dr. HARRIS said the third patient had improved considerably under treatment. The right pupil reacted fairly, he had recovered accommodation in the right eye, but the left remained the same. Then he had meningo-myelitis, and that, under mercury inunction and iodide, got well. Six years ago he had a fall—not a severe one—off a sofa, and jarred his back, and ever since then he had complained of symptoms in his back, numbness, pain, &c.: apparently a typical functional spine. He then developed the ocular and myelitic symptoms. With regard to the other cases, one sometimes met with pain in progressive muscular atrophy, certainly when there was joint trouble; and the pain here did not exclude poliomyelitis as a diagnosis. There had been no improvement in the woman's case. The man improved to a point, but only under strychnine. Neither of the cases gave a clinical history of syphilis, but in both the

Wassermann serum reaction was positive. In the man's case the infection probably occurred forty-two years ago.

Dr. GORDON HOLMES said he had made a post-mortem examination on a man who, at the time of death, presented many points similar to Dr. Harris's cases. He was a cab-driver, who twelve years previous to death had difficulty in raising luggage to the cab. He had slight pain, and came under observation four years later, when there was found to be almost complete atrophy of the deltoid, supra- and intra-spinati, and partial atrophy of the triceps. There was no sensory disturbance. The late Dr. Beevor examined him, and the diagnosis was left in doubt. The man became weaker, and walking was difficult, and he gradually developed spastic paraplegia. He died a few months after admission, from some other disease. He (Dr. Holmes) found the deltoid and the other muscles he had mentioned almost absent on one side, and on taking out the cord there was enormous thickening of the leptomeninges from the mid-dorsal to the second or third cervical segment. In places the dura was bound down to the leptomeninges, but was easily separated off. He had not yet fully examined the cord microscopically, but to the naked eye the cause of the muscular atrophy seemed evident, as the fifth and sixth anterior roots were much wasted on the side corresponding to the muscular wasting, and seriously constricted by the meninges as they left the dura mater. He asked Dr. Harris to say what he meant by the term syphilitic poliomyelitis. How was the lesion in the case to be correlated with the history of syphilitic infection, and how did syphilis produce that degeneration of cells? Was it primary, or a secondary disease of vessels? It would be interesting to collect these cases from the literature, to see how much evidence there was one way or the other.

Dr. PAGE MAY said all the cases which had been seen or described at that meeting had been treated by mercury; some by the mouth, others through the skin. No one had referred to the application of mercury by passing it, in a sterilized solution, directly into a vein. He had seen some cases treated abroad in which the intravenous injection of perchloride of mercury had apparently caused wonderful results in the way of rapid improvement. Dr. Head had emphasized the fact that mere rest caused marked benefit, even before the mercury applied through the skin could have had any effect. The direct injection into a vein should of course be carried out with full and definite precautions, but by this method the momentum and rapidity of action of the drug must be greatly increased, and prove of great value in numerous cases in which time is of urgent or vital importance. He asked whether members of the Section had considered that point.

Dr. SAVILL thought it doubtful whether Dr. Harris was justified in applying the term poliomyelitis to the cases, because, on clinical grounds, the symptoms of pain pointed rather more to a root or bone lesion. The presence of pain must indicate that the posterior roots were involved. Probably the disease began outside the cord.

Dr. HARRIS, in reply, said the diagnosis resolved itself into two heads: poliomyelitis and syphilis. The Wassermann reaction was positive, though there was no history of syphilis. Both had pathological conditions of the pupils. He admitted that the poliomyelitis was a little open to doubt, but the facts that there had been no sign of spasticity or of weakness of legs in either of the patients, that neither of them had anything approaching girdle pain or root pains, and that in the woman's case the pain was only slight in the left shoulder and absent on the right side, seemed to make it unlikely that it was a root lesion. Both had fibrillary tremors, in both the condition came on subacutely, and the woman showed the reaction of degeneration some months ago. It was difficult to fix syphilis on to the poliomyelitis. Autopsies had been described in similar conditions, and there was a good description of a case by Merle in the July number of the *Revue neurologique*.¹ The man had suffered from syphilis, and, as far as he could remember, he showed no syphilitic signs. The cord showed typical changes of extreme progressive muscular atrophy. It was a question whether all cases of progressive muscular atrophy were not due to some toxic lesion. When the history was inquired into, it was surprising how the "idiopathic" cases dwindled. If there was definite syphilis, was not one justified in assuming that that was the toxin which caused the lesion? That was why he had grouped those two cases, which resembled one another clinically, as probable syphilitic poliomyelitis.

Tabes with Amyotrophy.

By CECIL WALL, M.D.

J. M. B., MARRIED woman, aged 36. Previous history: Four years ago noticed loss of power in left thumb and first finger; the weakness gradually increased and began to affect left leg three years ago. About two years ago right knee became affected, gradually getting much weaker for last thirteen months. No previous illness. One child, aged 10; no miscarriages.

Present state.—Legs: knee-jerks not obtained; plantar reflexes not obtained; movement of legs generally weak, but no paralysis. Sensation: All forms seem dulled, worse in irregular patches; sensation of passive position good; wasting not very marked. Arms: Tendon reflexes not obtained; sensation fair to all forms of stimulus; sense of passive position good; both biceps and supinators active; loss of power of adduction of left thumb; weak grip; very little loss in right hand; left hand and forearm show fibrillary twitching; left hand, coarse tremors, worse on intentional movement; wasting of thenar and

¹ 1909, xvii, p. 877.

hypothenar muscles of both hands, more marked in left hand; no loss of power in arm or forearm of either side, though they seem wasted. Face: No paresis; no alteration in sensation. Respiratory system: Right side moves very little; breathing sounds very weak; bubbling râles all over. Pupils: Right larger than left; both react badly to light; both react on accommodation; fundi normal. Gait: Cannot stand alone; with help, ataxic, shuffling. Wassermann reaction positive.

Electrical reactions normal.

DISCUSSION.

Dr. WALL added that the radiograph showed that there might be a small cervical rib, and the question arose whether the wasting of the muscles of the hand was due to the cervical rib, or whether it was associated with change in the anterior horn of the spinal cord. One member suggested it might be simply due to occupation: the woman was a scrubber.

Dr. WILFRED HARRIS suggested it might be an occupation neuritis, rather than tabetic amyotrophy. The muscles mostly wasted were those of the thenar eminence on the left hand, the other intrinsic muscles of the hand being unaffected; and all those muscles would be related to the same segment of the cord. It was usual in cord affections to see the thenar eminence and the first interosseous go together. The woman worked hardest with the left hand, and that was the affected one; the trouble might be due to the pressure of the scrubbing brush on the thenar eminence. He had seen a similar condition produced by the use of a trowel, in a gardener.

Dr. WALL replied that he assumed that the Section looked upon the case as one of tabes with an occupation neuritis.

Spasmodic Neurosis. ? Paramyoclonus Multiplex.

By ARTHUR HALL, M.D. (Sheffield.)

A. L., AGED 32, single, shop assistant. Health good till March, 1906, when he had influenza; since then frequent attacks of "jumping" and "twitching" in the left shoulder. The attacks vary in severity, some being violent and very painful, as though the bones were being pulled out of their sockets, others being almost painless. They can be produced at any time by lifting up the arm, so that he has been obliged to stop using this arm for reaching things down in the shop; occasionally they come on without any apparent cause. They are always worse in

winter than in summer, at times disappearing altogether for days during the latter; an attack lasts about ten minutes. There is no history of any similar affection in other members of the family.

Present state: Tall man, with marked kyphosis of the spine; cranial nerves healthy; eye-grounds normal. No evidence of motor or sensory changes in limbs; reflexes normal and equal on two sides. Left arm: he holds it closer to the side than normal, and habitually avoids using it in order to prevent an attack; the left hand is colder than the right. There is some loss of muscular development, but not more than would be accounted for by disuse.

An attack is always developed by taking off his underclothes, unless he is very cautious. The attack begins by clonic contractions of the left pectoralis, often only in limited sections at a time; all the shoulder and arm muscles become involved, either in their entirety or in individual portions of the muscles. In a severe attack the scapula stands out from the back, and there is a certain amount of tonic spasm, whilst he suffers from severe pain in the shoulder. It usually passes off in five to ten minutes.

DISCUSSION.

Dr. FARQUHAR BUZZARD said Dr. Hall had been prevented from coming to the meeting, and had asked him to take charge of the case. Members would agree that it presented some unusual features. The attack, which was brought on by undressing and lifting the left arm to the top of his head, presented the following features: There were moderately severe contractions of parts of various muscles, chiefly the deltoid, the pectorals, the biceps, triceps, and other muscles attached to the scapula and humerus. There was nothing of the kind in the trapezius or muscles of the forearm. When any muscle was put into voluntary contraction, the spasmodic contraction of the muscle at once ceased. In abducting the shoulder against resistance, the greater part of the anterior and middle part of the deltoid ceased to show spasmodic contractions, while the posterior fibres, which were not concerned with the abduction, continued to jump in the way described. There had been no other signs of organic disease, and Dr. Hall would be glad to elicit opinions.

The CHAIRMAN (Dr. T. Buzzard) said he did not see the patient in an attack, but the description reminded him strongly of a case he saw several years ago in a man who had been in Africa with the Imperial Yeomanry, fighting and undergoing hardships. He had five or six attacks of shivering while there, probably malaria; and two or three more attacks on the voyage home. Later he developed attacks completely resembling those in the present case, and he (Dr. Buzzard) gave it as his opinion that it was paramyoclonus

multiplex. It was principally confined to the shoulder-girdle and neck muscles, the lower extremities being much less affected and the face not at all. His mother was highly nervous. When the man came to England and had treatment directed to his functional neurosis he gradually improved, and in a couple of years he had lost practically all the movements.

Thrombosis of Cerebral Arteries.

By HENRY HEAD, M.D., F.R.S.

P. K., AGED 27, ex-sailor in the Navy. In 1901 he suffered from lues (chancre). He was stationed at Chatham in 1906. On September 10 he woke with a left-sided headache, and by 10 a.m. he found he could not speak, but succeeded in getting to London. On his way his right arm became paralysed. The next day, September 11, 1906, he was admitted to the London Hospital. He was then entirely unable to speak, although he could read, write with his left hand, and name objects shown to him in writing. The right arm was weak, but the right leg was not affected. The lower part of the right half of the face was parietic. The tongue was protruded straight. The pupils acted well, and the disks were unaffected. The knee-jerks were normal and equal, and both plantar reflexes were of the flexor type. He was discharged on September 30 completely well.

On December 25, 1906, he had a drinking bout, and woke next morning to find he had lost power in the left leg and arm. He was admitted on December 27 with paresis of the left arm, the left leg, and both halves of the face. The left knee-jerk was greatly exaggerated, ankle clonus was obtained from the left foot, and the left plantar reflex was of the extensor type. The right knee-jerk was brisk, no ankle clonus was obtained, and the right great toe went downwards on testing the plantar reflex. He recovered, and was discharged on January 17, 1907, with some dragging of the left foot and paresis of left arm.

On September 24, 1907, he was taken ill suddenly whilst at work, and was re-admitted to hospital on September 28. He now had a complete left hemiplegia and complete motor aphasia. The optic disks were pink, but showed no definite swelling, nor were the edges blurred. The hemiplegia gradually cleared away, leaving slight weakness in the left arm and a slight dragging of the left foot. Speech, however, returned much less completely, and he still has difficulty in making

himself understood. The knee-jerks are brisk, the left greater than the right; ankle clonus is obtained from the left foot, but is absent on the right side. The left toe goes up, the right down, on testing the plantar reflex.

Cerebral Hæmorrhage from Luetic Vessels.

By HENRY HEAD, M.D., F.R.S.

W. M., AGED 32, engineer, in July, 1902, contracted gonorrhœa, which lasted about two weeks. This was followed by rash and sore throats. There is now a scar at the orifice of the meatus, pointing to the probability that the infection was syphilitic. In April, 1904, he felt dizzy, and within twenty minutes became paralysed down the right half of the body. He lost his speech, but did not become unconscious. Since that time he has shown no material alteration. He is still hemiplegic, his tongue is protruded to the right, the right knee-jerk and wrist-jerk are greatly exaggerated, ankle clonus is obtained, and the plantar reflex on this side gives an extensor response.

DISCUSSION.

Dr. HEAD said the two cases were brought to contrast the different effects which might be produced by a syphilitic vascular lesion of the brain within six years of infection. In July, 1902, W. M. had what he thought was gonorrhœa; this was obviously syphilis, as it was followed by a rash, sore throat, and there was now a scar at the orifice of the meatus. Treatment was very inefficient. One night, two years later, he felt dizzy and suddenly became hemiplegic, losing his speech. Speech gradually returned, but the hemiplegia had remained. Since he had been under observation he had had two courses of inunction with ung. hydrarg., with iodide in between, but the hemiplegia had not altered since April, 1904. This case was probably an instance of cerebral hæmorrhage, and, as in other cases of severe cerebral hæmorrhage, arising probably in the capsule, one would not expect that the condition would improve under treatment. The other man, now aged 27, was a sailor. In 1901 he suffered from syphilis (chancre). Treatment was perfunctory and intermittent. One morning (September 10, 1906) he awoke with left-sided headache; at 10 a.m. he could not speak, and gradually became paralysed in the right arm, with weakness of the right leg. Next day he was admitted, under Dr. Head's care, with right hemiplegia and total aphasia, but he could write his requirements and the names of things held up to him. Vigorous

inunction was carried out, and he left the hospital at the end of three weeks with no obvious defect. On Christmas Day, 1906, he had a drinking bout, and next morning awoke with the power gone from his left arm and leg. He was again admitted to the London Hospital and treated, and he was discharged on January 17 with dragging of the leg and paresis of the left arm. He was lost sight of until September, 1907, when he was again taken suddenly ill while at work, and was re-admitted next day. This time he had left hemiplegia, and, again, complete aphasia. He greatly improved, except in his speech. He was now engaged on the railway, but spoke very badly. In walking he dragged the foot a little, and the grasp by both hands was good. The response of the left toe was upwards, that of the right was doubtful. In hospital practice it was impossible to keep such patients under treatment, as they ceased to visit the hospital as soon as they thought themselves well. As a consequence the disease was seen far more in its natural form than in private practice, where patients permitted themselves to be treated in a proper manner.

Dr. HARRY CAMPBELL said it would be interesting to hear from Dr. Head the exact grounds on which he diagnosed hæmorrhage in the first case. From the data given such a diagnosis did not seem justifiable.

Dr. WILFRED HARRIS said his feeling was the same as Dr. Campbell's concerning hæmorrhage versus thrombosis. It was notoriously difficult to distinguish them, and the post-mortem records showed how often mistakes were made. Suddenness of onset was no proof that it was hæmorrhage. One man he remembered fell suddenly down as if he had a blow on the back of his head, and at the autopsy it was found to be thrombosis in the external capsule, catching the optic radiations and causing quadrantic hemianopia. The fact that Dr. Head's patient did not lose consciousness was rather in favour of thrombosis than of hæmorrhage, and so was the patient's age. He did not think the point could be settled clinically. It seemed to be largely a question of repair, and whether there was collateral circulation or not, and on the amount of the latter depended to a great extent the question of improvement. A cortical lesion might cause as much permanent paralysis as the present patient showed, and thrombosis was capable of producing permanent spasticity.

Dr. JAMES TAYLOR said there was another difficulty with reference to the second case. He supposed Dr. Head claimed that the improvement in the thrombotic case was due to the effects of treatment. But it seemed to him inconceivable, if there were a vessel thrombosed, even if it were syphilitic, that one could remove that thrombus by means of antisymphilitic treatment; and he thought that, even without the energetic treatment, if the man were at rest in hospital his improvement might have been as rapid and complete in the first instance as it was.

Congenital Lues causing Optic Atrophy and ultimately leading to Dementia Paralytica Juvenilis.

By HENRY HEAD, M.D., F.R.S.

E. L., FEMALE, aged 23. At 6 years old she went blind, and was sent to a school for the blind. Here she was taught to read and to make baskets. At the age of 20 she began to degenerate mentally, and developed epileptiform convulsions, which have gradually increased in frequency. She has now lost the power of reading; and although she can plait baskets, they are no longer of standard size, and the plaiting is irregular. She has become very excitable and emotional. Face and tongue are a little tremulous, the reflexes are brisk, but there is no paresis. Primary optic atrophy is present in both eyes. Her teeth show defective enamel, and there are scars at the angles of the mouth.

Chronic Syphilitic Meningitis with Compression of the Cord.

By T. GRAINGER STEWART, M.B.

A. B., AGED 64. Complaint: Weakness of the legs and stiffness.

Previous health: When aged 18 had syphilis; enjoyed good health till ten years ago, when he had a sudden seizure in the street and fell down unconscious. He was unconscious for a quarter of an hour, but had no paralysis after.

Present illness: Began gradually five years ago with stiffness and weakness of the right leg, and later of the left. This weakness has gradually become more marked, and at the present time he can walk with assistance and with the help of a stick. He has never had any sphincter trouble.

Present state: The pupils are both small and inactive to light; nothing abnormal above the fourth dorsal segment. Motor: Below this level there is a slight motor weakness, rather more on the right side than on the left, with relatively severe spasticity. Reflexes: The deep reflexes of the legs are increased with clonus. The plantar reflexes are extensor in type, and the superficial abdominal and epigastric are absent. Sensory: There is a slight diminution of all forms of sensibility below the level of the fourth dorsal segment. Spine: Normal.

Affection of Posterior Roots of Lower Cervical and Upper Dorsal Nerves? Tabes? Lateral Sclerosis.

By HARRY CAMPBELL, M.D.

C. J. S., AGED 42 years and 11 months. Syphilis twenty years ago; was treated six months. Eight years ago attacked with pain in back of neck; continuous; very severe "gnawing," "as if neck were fixed in vice," "gripping;" involved also the back as far as the ninth dorsal vertebra, and both upper arms. This lasted three to four months. Two and a-half years ago he was attacked with similar pains, which extended somewhat farther down the arms. For the last eighteen months there has been a steadily increasing numbness of his hands and inability to execute fine movement with them; often has not known where his hands were. For a somewhat longer time has complained of irritation—when clothes are on—in back of neck and front of chest. Diminished sexual power twelve months.

Present condition: Defective cutaneous sensibility (touch, pain, heat, cold) both upper extremities, especially in the region of the hands. Defective sensibility of ulnar nerves. Defective sensibility of the muscles of the upper extremities. Defective joint sensibility in the upper extremities. Jaw-jerk present; triceps-jerks brisk. On percussing any part of the circumference of the wrists, the fingers and thumbs flex. Cannot recognize objects placed in the hands without use of the eyes. Considerable inco-ordination of both upper extremities. Knee-jerks and ankle-jerks brisk. ? Babinski's sign present.

The diagnosis seems to point to a syphilitic meningitis involving the posterior roots of the lower cervical and upper dorsal nerves (? tabes) and to involvement of the lateral columns.

Neurological Section.

January 27, 1910.

Dr. J. A. ORMEROD, Vice-President of the Section, in the Chair.

Syphilis and Parasyphilis of the Nervous System.

By F. W. MOTT, M.D., F.R.S.

THE causal connexion of tabes dorsalis and general paralysis with syphilis is now so firmly established in the minds of most neurologists that it would be absurd, in dealing with such an important question as syphilis of the nervous system, not to include these parasyphilitic affections. Diseases of the nervous system due to syphilis may then be classified thus :—

(I) (a) *Syphilitic diseases of the brain*, comprising the following forms : Basic meningitis, meningitis of the convexity, cerebrospinal meningitis, arteritis, and, lastly, gummatous tumours. My experience, indicated by a large number of recorded cases in which autopsies were performed, shows that all these conditions may be more or less combined in the severe and early forms of the disease. (b) *Syphilitic diseases of the spinal cord*.

These diseases are all due to the direct effects of the syphilitic virus upon the mesoblastic nutrient, enclosing and supporting tissues of the central nervous system, causing secondary degenerative changes in the essential nervous elements.

(II) *Parasyphilitic affections* : (a) Tabes dorsalis ; (b) Tabes optica ; (c) Paralytic dementia.

In syphilitic disease of the nervous system we divide our cases into spinal and cerebral for clinical convenience, but my experience as a

pathologist tends to show that the whole cerebrospinal axis is usually, if not always, affected, although in some cases the spinal symptoms are the most obtrusive, and in others the cerebral; and that leads me to the consideration of the relation of syphilitic eruption of the skin to recognized and unrecognized syphilis of the meninges. Many cases of syphilis of the meninges could be cited where the symptoms have appeared within three months of the primary infection—e.g., one of the cases reported by Goldflam. A strong man, aged 40, one month after the appearance of an indurated chancre, was seized with violent pains in the neck, the shoulders, and upper limbs, with stiffness and immobility of the limbs. There was also a marked hyperæsthesia in the area supplied by the brachial plexus. The case was completely cured after twenty-four inunctions.

The following cases in my own practice are instructive: (1) A young man was sent to me complaining of headache, internal strabismus, and double vision; he had also commencing optic neuritis. I ascertained that ten weeks previously he had contracted a so-called soft sore, which had been treated only locally with iodoform. I put him on mercurial inunction, and the symptoms rapidly left him. (2) A man was admitted to Charing Cross Hospital, under my care, who, thirty years previously, had suffered with chancre, sore throat, and rash; and *was reinfected sixteen months ago*. He again had a chancre, rash, and sore throat. Within twelve months of infection he suffered with girdle pain on the left side, absence of abdominal reflex, and some root anæsthesia on this side, increase of the deep reflexes and slight Babinski on this side, together with inability to empty the bladder. He had a papular rash on admission, and general polyadenitis. There was a lymphocytosis of the cerebrospinal fluid not marked. He improved rapidly on mercurial inunction. The rash rapidly cleared up, and the local symptoms also. The case, therefore, was a localized syphilitic meningitis due to reinfection. This case is interesting, for it is rare to find a case of reinfection.

Sir William Gowers, in his Lettsomian Lectures, 1890, scathingly criticized the evidence brought forward by Long and others to show that a slight meningitis may be associated with the early phenomena of syphilis more frequently than was generally supposed. But the modern conception of the cause of the roseolar rash by the spirochæte of syphilis escaping from the blood in the cutaneous vessels makes it possible that a similar infection of the cerebrospinal meninges may occur.

The researches of Ravaut and others show the frequent existence of a lymphocytosis during the secondary period, sometimes very abundant, which proves there may be a meningeal reaction concomitant to the cutaneous eruption. A series of observations of lymphocytosis of the cerebrospinal fluid of early cases of syphilis, in which there occurred slight signs of meningeal irritation and a parallelism between the abundance of lymphocytes and the intensity of the signs of meningeal irritation, both disappearing and diminishing under treatment, would substantiate Long's hypothesis. Boidin and Pierre Weil point out that the study of the cerebrospinal fluid shows that the central nervous system is very frequently affected in the secondary period, but this affection may only be habitually shown by the cellular reaction of the meninges, and is sometimes associated with ocular troubles or paralysis of cranial nerves or other objective signs; it is exceptional, however, for signs of acute meningitis to recur. They describe the case of a young man, aged 18, admitted to the hospital with all the signs of an acute meningitis—headache, vomiting, constipation, rigidity of neck, Kernig's sign, inequality of pupils, and slight elevation of temperature. The first impression was that it was a case of tubercular meningitis, but upon examination of the patient the existence of an indurated chancre of the penis was discovered. No other specific sign was found; no mucous plaques, no roseola, no suboccipital adenopathy. Lumbar puncture revealed a pure lymphocytosis of average intensity; four days later the roseolar rash appeared. This patient rapidly recovered under treatment, and the following is a chronology of the symptoms: (1) Chancre, middle of June; (2) headache, July, 15; (3) signs of meningitis, August 5; (4) roseola, August 12; (5) cure of the meningitis, August 15.

The living organism in the blood presumably may, in rare instances, affect the blood-vessels of the meninges before the blood-vessels of the skin; occasionally the meninges are affected simultaneously with the skin, causing a sufficient degree of irritation to lead to manifest symptoms. But just the same as the roseolar rash may be so slight as to escape observation, so the eruption in the meninges may be so slight as to cause no symptoms; but if the meninges are infected, it is quite possible that the virus may remain latent until some other cause acts as a co-efficient in the production of a definite lesion with symptoms. The following case occurred in my practice and illustrates this point: A soldier, aged 28, contracted syphilis; eighteen months later he was thrown from his horse and he felt considerable pain in the back, but

was able to walk after the accident, and he observed no symptoms for a week; then he felt a tightness in the lower part of the abdomen, weakness in the legs, and difficulty with his bowels and bladder. He had been previously treated with mercury from the commencement of the chancre. I saw him eighteen months after the onset of the spinal symptoms. He had spastic gait, ankle clonus, Babinski's sign, difficulty with the bladder and bowels, abdominal and cremasteric reflexes absent; no loss of sensation. It is quite possible that an infection of the spinal meninges took place when the secondary eruption occurred, that it remained latent under the influence of the mercury, and that, owing to the temporary injury caused by the fall, the virus was able to decrease the resistance of the tissues and set up active proliferation. If once the virus has obtained access to the cerebrospinal fluid, it is difficult to dislodge; under mercurial treatment, as cases show, the most remarkable results may be obtained in the relief of symptoms, and patients may be completely cured and no remissions occur. I am not, however, so hopeful about the cure of brain syphilis as I was when I finished an investigation some ten years ago on forty cases of cerebral syphilis. I have had the opportunity of seeing what became of many of those cases that I believed were either cured or permanently relieved. I regret to say not a few are dead or have had serious relapses. Brain syphilis is certainly more serious than spinal syphilis; but, as I have said before, in the majority of instances there is an affection of the whole cerebrospinal axis found post mortem in fatal cases of syphilis of the nervous system, and I speak from a large experience.

THE INVESTIGATION OF A CASE OF SUSPECTED SYPHILIS OR PARASYPHILIS OF THE NERVOUS SYSTEM.

When a case presents itself we should ask ourselves and try to answer the following questions:—

- (1) Do the history, signs, and symptoms point to the disease being syphilis or parasyphilis of the nervous system?
- (2) Can any other cause than syphilis account for the symptoms?
- (3) Does lumbar puncture show the existence of lymphocytes in the cerebrospinal fluid, and, if so, are the lymphocytes in relative abundance?
- (4) Does the blood give the Wassermann reaction? Does the cerebrospinal fluid give the Wassermann reaction?

(5) What is the seat of the lesion as indicated by the signs and symptoms?

(6) Lastly and subsequently, does the result of treatment confirm our conclusions?

No doubt the clinical symptoms of syphilis of the nervous system will be fully considered by Dr. Head and others, who will take part in the discussion. I shall therefore limit my remarks especially to Questions 3 and 4.

EXAMINATION OF THE CEREBROSPINAL FLUID.

The normal fluid contains very few cellular elements, whereas in progressive parasyphilitic and syphilitic meningitic affections the lymphocytes are greatly increased in numbers. The amount of lymphocytosis is an index of the activity of the disease; it can also be used as an indication of the effect of antisyphilitic treatment. I have observed the lymphocytes diminish considerably and the signs and symptoms of the disease diminish correspondingly in cases of syphilitic meningitis.

Lymphocytosis in tabes and general paralysis does not diminish with antisyphilitic treatment, and this method is therefore useful in differentiating cases of pseudo-tabes or pseudo-general paralysis, both of which may considerably improve with antisyphilitic treatment. But lymphocytosis occurs in other affections in which syphilis and even meningitis play no part. It cannot therefore be regarded as absolutely diagnostic of a meningitis, but it is strong presumptive evidence, and, when combined with other facts, the existence of a lymphocytosis of the cerebrospinal fluid is an invaluable sign of the syphilitic and parasyphilitic affections. Particularly is it useful in deciding between a case of early doubtful general paralysis and other troubles—e.g., a patient suffering with neurasthenia and alcoholism.

In general paralysis, and to a less degree in tabes, there is also an increase in proteid in the cerebrospinal fluid. If simultaneously hæmolytic, lymphocytic, and albumin diagnostic examinations be made in the same samples of cerebrospinal fluid from the same patient, there will be found fluctuations in the degree of manifestation of their reactions, but they will always, or nearly always, be present.

The Wassermann Reaction.

In the hands of nearly all trustworthy and experienced investigators this method, introduced by Wassermann, has yielded most valuable results as a means of diagnosis. It is even claimed that for general paralysis it is more reliable than the Widal reaction for typhoid. According to Plaut, the reaction may be negative with the cerebrospinal fluid in cases of syphilis of the nervous system, but he obtained a positive result in 94 out of 95 cases of general paralysis with the cerebrospinal fluid, and in every one of the cases the serum gave a positive reaction. In cases of cerebral syphilis the serum was usually positive and the cerebrospinal fluid usually negative.

At my suggestion Dr. Henderson Smith, of the Lister Institute, and my assistant, Dr. Candler, recently examined by the Wassermann test the cerebrospinal fluid of 127 cases of various forms of insanity. Of this number, 64 were cases of general paralysis, and in 59—or 92.1 per cent.—a positive result was obtained. Of these 59 cases, 21 have since died, and the clinical diagnosis of general paralysis has been confirmed by the post-mortem investigation. Fluids from 63 cases not suffering from general paralysis were also examined, and in no single instance was a positive reaction obtained. A few of these cases have since died, but none showed at the post-mortem examination any evidence of general paralysis. Seventeen out of the 21 cases of general paralysis above referred to, which came to the post-mortem table, showed in each case before death an excess of lymphocytes in the cerebrospinal fluid. Marie and Levaditi have stated that a positive reaction by the Wassermann method does not necessarily accompany an excess of lymphocytes in the fluid, and in 5 cases in the above series of investigations, in which the Wassermann reaction was negative, there was an excess of lymphocytes in the cerebrospinal fluid. None of these patients has yet died, so that the diagnosis of general paralysis has not been absolutely confirmed. On the other hand, in 17 out of the 21 cases of general paralysis, which were verified by post-mortem investigation, the cerebrospinal fluid was examined during life, and showed an excess of lymphocytes, together with a positive Wassermann reaction.

Concerning the chemistry of the Wassermann reaction, I have found that a fluid giving a positive reaction fails to do so after the separation of the protein fraction. Noguchi has since come to the conclusion that the substance in the fluid causing the reaction is attached to the

euglobulin, from which it cannot be separated by solvents. Concerning the value of this reaction, I will give three striking examples out of many:—

(1) I recently saw a woman in one of the London County Asylums with double optic neuritis, vomiting, and headache. I had her transferred to my care at Charing Cross Hospital. Mr. Collins found five diopters of swelling in each disk. The cerebrospinal fluid did not give the Wassermann reaction, but contained abundance of lymphocytes. She was put upon mercurial inunction. The swelling of the disks rapidly subsided, and from being unable to read large print she was able to read small, the headache ceased, and the vomiting no longer occurred. She was subsequently discharged apparently cured.

(2) A man was admitted to one of the London asylums; he improved so much that the medical officers considered it doubtful whether he was a general paralytic. Lumbar puncture was performed, and the fluid was examined by the Wassermann test; it was positive. I expressed the opinion that it was certainly a case of general paralysis. He still continued to improve, and even his discharge was contemplated. I maintained that the test was not likely to be wrong; the next time I visited the asylum my prediction was confirmed. He had had several seizures and within three months he was dead, and examination of the brain left no doubt about the correctness of the diagnosis.

(3) A woman, aged 34, was admitted to Charing Cross Hospital under my care, said to be suffering from tabes. There were no signs of syphilis on the body. Her youngest child is aged 4. Fifteen months ago she had a seven months' stillborn infant. Four months ago she suffered with numbness in the legs, of which she took little notice; then she had double-vision and tingling in the feet and legs. For the past fourteen days she has suffered with a girdle sensation. She now complains of lancinating pains extending from the back down both legs, unsteadiness in gait and station, a feeling of the soles as if walking on cork, unequal pupils which reacted sluggishly to light and to accommodation, pain and cramp in the muscles of the legs, absent knee-jerks, patches of anæsthesia on the legs, and a belt of thoracic anæsthesia with girdle sensation. After inquiring into the history and finding that she had suffered with headache and squint, that the knee-jerks—which were absent on admission—had returned a few days later, I came to the conclusion that this woman, with a probable duration of infection of less than four years, was suffering really from pseudo-tabes, the result of syphilitic meningitis, especially as she told me that she had suffered

with a little stiffness of the neck, and I then obtained Kernig's sign. Lumbar puncture was performed, and 390 lymphocytes per c.mm. were found—an enormous number for tabes dorsalis. This large number of lymphocytes could only be accounted for by a widespread, active, gummatous meningitis. She was placed on mercurial inunction, and a fortnight later the lymphocytes had fallen to 70 per c.mm., and the fluid this time was tested by the Wassermann method and found to give a negative result. Unfortunately the blood was not tested on this or future occasions. A fortnight later the cerebrospinal fluid was examined and only 20 lymphocytes per c.mm. found, the patient being almost well. A fortnight later there were still no lymphocytes and the fluid was negative to the reaction. The patient was quite well; the pains, anæsthesia, and unsteadiness had entirely disappeared. She is still quite well, and twelve months have elapsed, but there is no guarantee that this woman may not have a recrudescence of symptoms; for my experience, clinical and pathological, has taught me that if once the contagion invades the subarachnoid space, producing a diffuse meningitis, symptoms of a latent affection becoming once more active may occur at any period after.

The case I have described shows the desirability of trying a mercurial cure in all cases of tabes and general paralysis in which there are atypical characters, where the Wassermann reaction is not obtained when the cerebrospinal fluid is examined and the lymphocyte reaction is marked. As clinical indications of pseudo-tabic lesions may be mentioned the following symptoms: (1) Sudden onset, or comparatively sudden onset, and rapid progress of symptoms; (2) Early appearance of affection after primary infection; (3) A variability in the condition of the tendon reflexes, especially patellar, and Achillis reflexes at one time lost, at another present, even increased and more marked on one side than the other; (4) The optic-nerve lesion causing a unilateral central scotoma, the other eye unaffected; (5) The marked improvement under treatment.

PARASYPHILITIC AFFECTIONS: HOW ARE THEY CAUSED?

Since the discovery of the *Spirochæta pallida* and the widespread practical application of the Wassermann reaction to the diagnosis of syphilis and parasyphilis of the nervous system, new interest has been added to the problem of the pathogenesis of tabes dorsalis and general paralysis. The ætiology of these two diseases is identical: the most

characteristic physical sign—the Argyll-Robertson pupil—is common to both diseases, and is, practically speaking, met with in no other diseases. All the evidence of the ætiology of tabes and general paralysis tends to prove that there is probably one essential cause—syphilis acquired or congenital—and that there are a number of co-efficients, any one of which, by itself or in combination with others, is not capable of producing the disease. The fact that congenital syphilis leads both to tabes and general paralysis at so early a period of life as to exclude most of the contributory factors, except neuropathic heredity, is an argument in favour of syphilis being the essential cause. Moreover, since the sexes are equally affected with congenital syphilis, so males and females are affected in about equal numbers with juvenile paralysis or tabes.

Although syphilis is the essential cause, yet, as Fournier showed, these diseases are not really syphilitic, but an outcome of syphilis, and the riddle is still unsolved why only about 3·5 per cent. of the persons infected with syphilis should subsequently suffer with one of these degenerations of the nervous system, termed “Parasyphilis.” But only 15 per cent. of persons suffering with diphtheria develop post-diphtheritic paralysis. These are usually cases in which the local infective process was mild and often unnoticed; in that respect, therefore, like parasyphilitic affections, which more often than not follow mild and even unrecognized primary infection and secondary symptoms. Is it because the virus is attenuated or modified, and thereby has acquired a special neurotoxic action, or is it because in a small percentage of individuals the cells of the body, *especially the cells of the nervous system*, react to the virus in a hypersensitive manner? As already indicated, there are facts which suggest the possibility of a certain form of virus with a neurotoxic action. Thus Babinski remarks that it seems possible that a syphilitic virus may sometimes be endowed with a particular aptitude for attacking the nervous system. He reports the case of two students who were infected the same day by the same woman; both died fifteen years later of general paralysis; these students were, however, related. I have recently heard of two professional men, not related, who acquired syphilis about the same time from the same nurse; ten years later they developed general paralysis. Marie and Bernard relate the instance of two men who were infected from the same source, and ten years later suffered with tabes. Erb narrates an instance of four patients infected by the same woman, who later became the subjects of either tabes or general paralysis; whilst a fifth, who had connexion with the woman but was not infected, did not suffer from any disease later. I

am indebted to my friend Dr. George Gibson for calling my attention to the following striking example given by Morel-Lavallée :—

Marthe X.				
May, 1870, mistress of <i>Primus</i> (?) (Medical Student), and gave him syphilis. He died, 1873, of syphilitic meningitis.	December, 1871, mistress of <i>Secundus</i> (Medical Student), to whom she gave syphilis. He married later, had two healthy children, and died, 1888, of general paralysis.	January, 1872, lived four years with <i>Tertius</i> (Medical Student). He married later, had two healthy children, and died, 1882, of general paralysis.	Later, mistress of <i>Quartus</i> (Chemist). He died, 1890, of general paralysis.	Still later, mistress of <i>Quintus</i> (Engineer). He died (no date) of folie syphilitique.

Probably the most striking example supporting this theory of a special neurotoxic virus has been afforded by Brosius, who relates that seven glass-blowers suffered with chancre of the lip, and out of five who ten years later came under observation, four suffered with either tabes or general paralysis. If we accept the fact that a spirochæte is the specific causal agent of syphilis, it is conceivable that there may be varieties of this organism as there are of the malarial parasite or trypanosome. Again, the organism may become modified in its passage through the bodies of certain individuals, or it may be modified by the action of mercury. It may thus happen that the virus may vary in different cases of infection. This, however, is speculation, and not only is not supported but also is rather contraindicated so far by experiments on animals. For, although lower apes have the disease in a mild form when inoculated from the human being, yet the syphilitic virus of an infected *Macacus rhesus*, when used to infect a chimpanzee, appears to have lost none of its original virulence, for the chimpanzee suffers as badly as if it had been infected direct from the human source of the virus. We are probably therefore on more certain grounds in attributing the variations of the effects which will follow infection not to the variation of the virus but to the reaction of the individual himself; and we may represent this in the form of an equation :—

$$\text{Symptom complex } x = \frac{V}{R} = \frac{\text{virus}}{\text{resistance.}}$$

If the virus V is constant, R resistance must vary. But R is made up of a number of factors, some of which we can ascertain, but it is

generally impossible to decompose *R* into all its constituents. Roughly speaking, we may say that it is made up of what a man is born with, what has happened after birth, and what will happen in the future to resist the action of the specific virus, which in the majority of instances is of lifelong duration. Most authorities agree that with the widespread syphilization of a race for many generations, the disease tends to assume a milder form; the effects of the disease are not so severe, and a widespread tendency to an inherited immunity has been brought about. The conversion of a rural into an urban population has done much towards racial syphilization and to the diffusion of a tendency to inherited immunity and the begetting thus of a mild form of the disease. But whereas there are fewer cases of severe syphilis than formerly, there are more cases of tabes and general paralysis. The interesting description given by Col. Lambkin¹ of the syphilization of the natives of Uganda shows how severely a race previously free from this disease suffers from malignant skin, bone, and visceral disease. He also points out that parasyphilitic affections are rare, the reason being that the disease has not existed in the country for a sufficiently long time to allow of their frequent occurrence. If we consider some facts concerning congenital syphilis we must come to the conclusion that immunity is possible; how otherwise can we explain the law of Profeta—viz., that the non-syphilitic child of a syphilitic mother does not acquire syphilis from the syphilized mother who suckles it? Again, the child may be syphilitic and the mother show no sign of syphilis; nevertheless the mother does not acquire syphilis by suckling that syphilitic child, whereas a wet nurse does. In the former case the foetus has acquired some antitoxin or something from the maternal blood which has stimulated its own tissues to react against the virus; in the latter (Colles's law) the mother has derived from the blood of the syphilized child an antitoxin or something (not the living contagium) which has stimulated her tissues to react against the virus so effectively that she cannot be infected. There is no reason to suppose that the germ cells do not participate in this reaction, seeing that every cell in the body is subjected to the sensitizing influence of the chemical products of the virus by means of the blood and lymph.

The histories I obtained in a large number of cases of juvenile general paralysis and cases of congenital syphilitic nervous disease revealed the fact that the mother very frequently had miscarriages, abortions, and typically syphilitic children without herself suffering at

¹ "System of Syphilis," ii, pp. 339-355.

all or presenting any signs of syphilis. In two instances the mother died of general paralysis; in a considerable number of instances the father died of this disease. As a general rule the history obtained from the parents of juvenile paralytics is as follows: miscarriages, abortions, dead children, children dying in infancy, often of meningitis or hydrocephalus, children who later in life suffered with nervous affections—e.g., nerve deafness, paralytic dementia, optic atrophy and tabes—and, finally, healthy children; and such a chain of circumstances would undoubtedly indicate that either the virus was becoming attenuated or the resistance to its action had been increased. In any case we may suppose that the children who were born with a syphilitic rash would be immune to reinfection, also those who afterwards suffered with parasyphilis; Krafft-Ebing's observations support this premiss. It is probably a question of degree of immunity to reinfection that would obtain in the presumably healthy children that followed the diseased ones. But this chain of events does not always occur, for sometimes children may be born with signs of heredo-syphilis after the birth of several healthy children, also parasyphilitic children may be born after the birth of several healthy children. This may be explained by the fact that the specific virus has become active again in the mother, which inference is negated in most instances by the fact that she herself may say that she has been in good health and no signs of the disease can be discovered in her. Another explanation offers itself, and that is that the specific virus may have attacked one ovum and spared another. Levaditi has seen the spirochæte within an ovum. No two individuals, even of the same family, are born alike, because the germ plasm out of which they were formed may be similar, but is not the same; one inherits certain ancestral tendencies which the other does not; and it may happen, therefore, that a child born later than the healthy children possesses less inborn resistance to the action of the virus; and consequently manifests congenital syphilis, or later parasyphilis. How can we explain this process of decay of particular groups, systems, and communities of neurones? Why should we have optic atrophy in one individual, atrophy of the spinal portion of the sensory protoneurones in another, decay and atrophy of the cortical neurones in a third, and in many instances a decay and atrophy of the whole nervous system. We cannot suppose that it is caused by the random metastasis of the syphilitic organism in the membranes, or coats of the blood-vessels, conveyed by the lymph or blood-stream, as is probably the case in the true syphilitic lesions of the brain and spinal cord. Everything points against this, for, although parasyphilitic

affections present the most varied signs and symptoms, there is one sign usually present which is for all practical purposes only met with in parasyphilis—viz., the Argyll-Robertson pupil. No coarse random lesion will explain the constancy of this phenomenon; moreover, this condition, although a sign of syphilitic infection, does not occur in true syphilitic brain disease. Spirochætes have never been found in the cerebrospinal fluid nor antigens. Antibodies are found in abundance and probably proportional to the extent of neuronie decay in tabes and general paralysis. I think all the facts are against the views of Lesser, Bosc, Hirschl, and others, that these late manifestations of degeneration of the nervous system may be regarded as quaternary syphilis, a very late effect of the virus comparable with syphilitic orchitis, glossitis, and other sclerosing lesions. According to this view we should be compelled to consider the meningeal and perivascular infiltrations and the glia-cell proliferation as the cause of the degeneration. But there are many reasons why we cannot accept this hypothesis. The view I take of the process is that parasyphilitic disease of the nervous system depends upon two factors, intrinsic-innate, and extrinsic-acquired—the soil and the seed; the vital resistance and the specificity of the virus, $\frac{V}{R}$. All those conditions which may be inherited or acquired, and which tend to active metabolism of systems, communities, and groups of neurones functionally correlated, and which, owing to those conditions of stress which in one individual would cause spinal neurasthenia, in another cerebral neurasthenia, will, in conjunction with the stimulating effect of the syphilitic poison, cause the nerve cells to exercise an abnormal metabolic activity in the production of the side-chain molecules necessary for immunization against the toxic effects of the virus.

Ehrlich points out that we cannot suppose that the cells of the body possess *per se* an executive defensive capacity to neutralize the noxious effects of all forms of organisms, and his work on hæmolysins show that the hæmolysin for the corpuscles of a particular animal only occurs after incorporation of the molecules of those corpuscles. But we may suppose that there is an *inherent* aptitude for the cells of the body of certain individuals to readily adapt themselves to defence against the action of the syphilitic virus in a race that has been widely syphilized for generations; consequently a larger number will have a mild form of the disease.

The nerve cells are perpetual elements incapable of regeneration, highly differentiated and complex in structure and function; their centre of nutrition is the nucleus, and, when decay sets in, the retrogressive

process attacks first the fine twigs and branches and rootlets of the tree, the dendrites and dendrons; in fact, the process is an inversion of its growth and development. But what should cause this premature decay and lack of durability? For the specific energy of the whole of the neurones in the healthy body is sufficient to last until the vital spark dies out. We know the prolonged duration of infectivity of the syphilitic virus as compared with other contagious diseases, also that one attack of syphilis confers immunity during the rest of the individual's life; moreover, the experiments of Krafft-Ebing are important to remember in this respect. I may remind you that Krafft-Ebing inoculated nine general paralytics with the virus of a hard chancre, and watched them one hundred and eighty days. Not one of these developed any signs of infection, although the cases selected were those in which there was no proof of antecedent syphilitic infection. The nerve elements being perpetual, and having acquired a habit of throwing off side-chain molecules, will continue to do so during life, and will contribute largely to the immunity produced. When there is no longer metabolic equilibrium, and decay sets in, these antibodies are thrown off in increasing numbers; this seems probable from the fact that in general paralysis and tabes the quantities increase with the progress of the decay. The process of decay will manifest itself in the earliest stages by an increased irritability and functional activity of the nervous structures, often manifesting itself in a *hyperæsthesia sexualis*, and not infrequently in striking intellectual activity, followed in each case by exhaustion and loss of function.

To follow the argument further it is necessary to explain the meaning of the term "lipoid substances." They are found in all animal and vegetable cells, and are probably as important for the vital activities of protoplasm as the proteins themselves. They consist of three groups: (1) Nitrogen and phosphorus free—viz., cholesterin, fatty acids, and lipochromes; (2) cerebrosides, bodies containing nitrogen but no phosphorus, phrenosin, and kerasin; (3) phosphatides (lecithins) containing both phosphorus and nitrogen. It is probable that the cytase or complement which leads to hæmolysis in the presence of the amboceptor acts by virtue of a ferment (lipolytic) action upon the lipoid substance of cells whereby they become unloosened and liberated into the blood. It is possible that the abundant presence of these lipoids in the blood and cerebrospinal fluid may account for the exaltation and excitement so characteristic of general paralysis. Again, it is possible that the toxic effects of bacterial poisons from secondary and terminal infections may be greatly increased by the presence of these lipoid substances.

The uselessness of antisyphilitic remedies is thus easily accounted for; indeed, they are generally positively injurious in true tabes and general paralysis, because they lower the vital energy in a system which is hypersensitive to the syphilitic virus. The only hope of doing any good is by an early diagnosis of the disease and suppression of all those exciting causes which use up the nervous energy and tend to overturn the normal metabolic equilibrium of the nervous structures. Other factors come in determining the location of the degeneration, and although microbial infections and microbial toxæmias are not directly responsible for these parasyphilitic affections, yet they may be an exciting agent to the onset of the disease, the aggravation of the symptoms, and the acceleration of the progress of neural decay.

I have often observed when influenza, dysentery, or pneumonia was prevalent in the asylums that a number of general paralytics died after a succession of epileptiform or apoplectiform seizures, and I have found post mortem that they were suffering from one of these morbid infections. Bacterial invasion, secondary or terminal infection of the organs of the body of a non-specific nature, frequently accelerate the morbid process of decay and bring about the fatal termination.

Syphilis of the Nervous System within Six Years of Infection.

By HENRY HEAD, M.D., F.R.S.

THERE is scarcely any disease which so directly expresses the pathological changes by which it is produced as syphilis of the nervous system. These changes can be reduced to three: (1) Disease of the vessels, which may lead to hæmorrhage or to secondary necrotic changes not in themselves syphilitic; (2) Chronic hyperplastic inflammation; (3) Syphilitic neoplasms.

When we attempt to classify and describe the clinical forms produced by these changes, it is essential to bear in mind that no one part of the nervous system is affected alone; nor is any one process ever alone at work. Within six years of infection, however local may be its clinical manifestations, the disease is widely diffused throughout the nervous system. Hence, in the majority of cases there is some unexpected, some bizarre element which at once suggests syphilis, such as a

hemiplegia with facial paralysis of the peripheral type or with radicular sensory changes (Case XVI).

I have gathered together thirty-three cases of men, seen by myself, in whom some nervous manifestations appeared within six years of infection. I have rejected all those where the date of infection was unknown, and have included those only in whom the primary and secondary manifestations were distinct enough to make the diagnosis certain.

The average date for the onset of nervous manifestations was two years and eight months after infection; but five cases occurred within one year. Amongst them the earliest period at which definite signs were discovered was three months; but this patient had suffered from continuous headache since the first development of his chancre.

PRODROMAL SYMPTOMS.

In a large proportion the onset of gross manifestations is preceded by headache—intense, widespread, and usually worst at night. Sometimes it is accompanied by nausea, but vomiting is uncommon at this stage. Sleeplessness is a frequent symptom. The man becomes changed. A doctor forgot how to do his morning round, and came back to his house after visiting each patient. In one case, I was told that the ship had been really without a first mate, so useless did a bright young officer become in this stage. The patient becomes irritable, morose or lethargic, lying long in bed and neglecting his work (Case I).

In this stage root-pains attributed to rheumatism or to sciatica are not uncommon. If these symptoms lead to a correct diagnosis and vigorous treatment, they may pass away entirely, to revive later before the onset of the nervous manifestations which throw the patient into the hands of the neurologist.

CEREBRAL SYPHILIS.

I shall not weary you by enumerating the various isolated symptoms and signs produced by syphilis of the brain; for since in every case both vessels and meninges suffer to a greater or less extent, there is scarcely a symptom or sign that may not appear at one time or another. It is by the complete picture, and not by any single detail, that the syphilitic nature of the malady can be suspected. Let me give you shortly a few pictures of the form assumed clinically by cerebral syphilis.

A young man, aged 21 (Case I), who had contracted syphilis four years before, was brought to me in August, 1908, in a highly exalted

condition. In March, 1908, he had become lethargic and would not work. But in June he became exalted; he bought motor-cars for which he could not pay and careered about the country at night, behaving in an altogether spendthrift manner. I could find no signs of gross disease beyond an ulceration of his nasal septum, obviously syphilitic. Before arrangements could be made for effective treatment, he was seized with a series of epileptiform convulsions. He became so violent and difficult to manage that he was transferred to an asylum. In four months he was discharged, but in March, 1909, had two severe fits. I lost sight of him until December, 1909. He then had two more fits, preceded by some months of progressive stupidity.

Case I.—In 1904, at the age of 17, a bad attack of syphilis, which was treated intermittently. In 1907 his nose began to ulcerate; no treatment.

In March, 1908, became depressed and would not speak; would not work; lay long in bed. In June, 1908, became exalted. Was out all night, riding about the country in motor-cars he had hired but did not pay for. Sexually greatly excited. Seen by me August 10, 1908. Statements rational, but in no way corresponded with facts; morose in manner. No abnormal physical signs except the ulcerated nasal septum. August 13: Series of epileptiform convulsions; violent; sent to asylum. December 10, 1908: Discharged cured. March 5, 1909: Two severe fits from which he recovered, and his parents refused to have him treated. September, 1909: Began to again become slow and stupid. Seen by me again, December 5, after another series of convulsions; very lethargic; memory defective. No abnormal physical signs; disks clear. Great improvement under vigorous inunction and rest in bed.

This case corresponds closely to that of a patient (Case II) who first came under my care in 1900, and finally died in Claybury Asylum in 1908. Dr. Mott's examination of his brain showed old syphilitic meningitis with universal endarteritis.

Case II.—In 1898, at the age of 24, severe attack of syphilis, for which he was treated very irregularly.

In 1900 seen by me for severe headache and inability to work; lay all day in bed. Memory defective. Treated with mercury, and improved. Admitted to hospital November 10, 1901, with the history that for the last few months he had had epileptic attacks. Intelligence had been growing worse; much headache. Left eyelid had drooped. On admission he was semi-conscious and violent; delirious. Rapidly recovered with rest in bed and inunction of mercury. All memory lost for recent events. Left the hospital apparently normal. No physical signs; disks normal. During the next six years I saw him twice, but he carried out no regular treatment and ceased to attend after a few weeks. June 25, 1908: Readmitted to hospital, demented, violent, wet and dirty. No abnormal physical signs. Died in

Claybury Asylum, July 3, 1908. Post mortem by Dr. Mott: Old syphilitic meningitis and universal endarteritis of the cerebral vessels.

Some of these patients become slowly demented with transient ophthalmoplegia or hemiparesis, and yet, as in Case III, may recover completely.

Case III.—In August, 1901, at the age of 21, whilst serving in South Africa, developed a chancre. Was treated for two and a half years with mercury in pills, which he took regularly.

November, 1905: Developed headache and vomiting, which passed away. February, 1906: Headache and vomiting returned, with vertigo. May, 1906: "Stroke"; speech became impossible. Similar attack five days later. October, 1906: Another attack; right half of his face became paralysed; could not write. From that date he steadily degenerated, and when I saw him in June, 1907, he was extremely demented. All memory was gone; he did not know he had served in the war. Could not tell the year, and muddled up the few facts he still remembered of his illness. Pupils reacted to light, and, although he complained of attacks of double vision, there was no ophthalmoplegia. Disks normal; no abnormal physical signs. During the next two and a half years he had four courses of inunction, with massage and rest in bed. He is now absolutely normal, and has recently passed an examination for promotion.

A schoolmaster under my care was suddenly attacked with aphasia four and three-quarter years after infection (Case IV). This passed off slowly, but recurred in similar attacks, preceded by violent headache and accompanied on one occasion by numbness of his right leg. I could find no gross signs, but the condition passed away under treatment with mercury and iodide.

Case IV.—In March, 1903, at the age of 25, chancre. Eight months later, rash; chronic sore throats. For three years was treated by a well-known specialist with mercury in pills. In 1907 developed a syphilide of the face, for which he was again given mercury.

January, 1908: Sudden difficulty in speaking, which rapidly passed off. June, 1908: Violent headache and vomiting. August 9, 12, and 16: Sudden difficulty in speaking, the last attack accompanied by pins and needles in right leg. August 23: Prolonged loss of speech with a peculiar sensation all down right half of body. September, 1908: I could find no abnormal signs except that both knee-jerks were brisk. No Babinski's sign. Disks normal.

True epileptiform convulsions may occur even three years after infection, and are frequently difficult to treat (Case V).

Case V.—In 1897, at the age of 26, developed a chancre. Repeated sore throats. Was treated for eighteen months with mercury by the mouth in pills.

In 1900 he began to have attacks in which he became momentarily unconscious; these did not interfere materially with his work. In February, 1906, he had his first major attack; since then several such attacks, which were always preceded by hours or days of violent headache. Jaw goes over to the right and he then loses his senses. Seen by me in 1907. I could find no abnormal signs in the nervous system; disks were normal. He was depressed but not stupid, and was able to carry on his work as a school-master.

Hemiplegia, with or without aphasia, is a frequent result of syphilis within six years of infection; in one of my cases it occurred within eighteen months of the primary sore (Case VI).

Case VI.—In October, 1906, at the age of 27, he developed a chancre, followed by sore throats and a rash. He was treated continuously with mercury, by the mouth, up till September, 1908.

April 15, 1908: Numbness of the right arm and leg, followed the same night by a fit and right hemiplegia. Seen by me on September 23, 1908, for right hemiparesis, including face and tongue; irritable, could not find his words; right knee-jerk greater than left, plantar reflexes flexor; no rigidity. October 5, 1908: Whilst under treatment with mercury had a violent convulsive attack, developed total left hemiplegia, and died on October 7 of exhaustion.

These hemiplegias differ greatly in their mode of onset and subsequent behaviour. Usually the patient complains of numbness and gradual loss of power, which, as in Case VII, becomes a complete hemiplegia in about twenty-four hours; or, as in Case VIII, the onset may be sudden and the paralysis may be complete within a few minutes.

In three of my cases where the onset was sudden, it occurred within two years of infection; but, in spite of energetic treatment, none of them improved materially. On the other hand, those hemiplegias which come on slowly yield to treatment in a remarkable manner.

Case VII.—Ex-sailor in the Navy. In 1901, at the age of 21, he suffered from syphilis (chancre).

He was stationed at Chatham in 1906. On September 10 he woke with a left-sided headache, and by 10 a.m. he found he could not speak, but succeeded in getting to London. On his way his right arm became paralysed. The next day, September 11, 1906, he was admitted to the London Hospital. He was then entirely unable to speak, although he could read, write with his left hand, and name objects shown to him in writing. The right arm was weak, but the right leg was not affected. The lower part of the right half of the face was paretic. The tongue was protruded straight. The pupils acted well, and the disks were unaffected. The knee-jerks were normal and equal, and both plantar reflexes were of the flexor type. He was discharged on September 30 completely well. On December 25, 1906, he had a drinking bout, and woke next morning

to find he had lost power in the left leg and arm. He was admitted on December 27 with paresis of the left arm, the left leg, and both sides of the face. The left knee-jerk was greatly exaggerated, ankle clonus was obtained from the left foot, and the left plantar reflex was of the extensor type. The right knee-jerk was brisk, no ankle clonus was obtained, and the right great toe went downwards on testing the plantar reflex. He recovered, and was discharged on January 17, 1907, with some dragging of the left foot and paresis of left arm. On September 24, 1907, he was taken ill suddenly whilst at work, and was re-admitted to hospital on September 28. He now had a complete left hemiplegia and complete motor aphasia. The optic disks were pink, but showed no definite swelling, nor were the edges blurred. The hemiplegia gradually cleared away, leaving slight weakness in the left arm and a slight dragging of the left foot. Speech, however, returned much less completely, and he still has difficulty in making himself understood. The knee-jerks are brisk, the left greater than the right; ankle clonus is obtained from the left foot, but is absent on the right side. The left toe goes up, the right down, on testing the plantar reflex.

Case VIII.—In July, 1902, at the age of 28, he contracted gonorrhœa, which lasted about two weeks. This was followed by rash and sore throats. There is still a scar at the orifice of the meatus.

In April, 1904, he felt dizzy, and within twenty minutes became paralysed down the right half of the body. He lost his speech, but did not become unconscious. Since that time he has shown no material alteration. He is still hemiplegic, his tongue is protruded to the right, the right knee-jerk and wrist-jerk are greatly exaggerated, ankle clonus is obtained, and the plantar reflex on this side gives an extensor response.

I have once seen pure apraxia (*Case IX*) affecting the right half of the body within four and a half years of infection. The patient had been vigorously treated by the mouth for three years. A year later he developed enlarged glands and was again given mercury by the mouth. Shortly afterwards he slipped and fell, whether in consequence of a seizure or an accident it was impossible to say; he insisted that he trod on a woman's dress when going down the steps. Next morning he went to sleep in his office, and a little later began to dictate nonsense. Next day he was profoundly apraxic. This condition cleared up entirely.

Case IX.—In January, 1905, at the age of 35, he developed a chancre. For six months he suffered with sore throats and developed an erythematous and papular rash. He took mercury by the mouth in pills for three years, under the guidance of a specialist. June, 1909: Developed some enlarged and tender glands and was again put on to mercury by the mouth.

Case. August 11, 1909: Fell down the steps at a station—he says he tripped over a woman's dress; he was quite well that night except for headache. August 12: Took pills. Went to the city, fell asleep in his office, and after lunch dictated nonsense;

was sent home; slept well. August 13: Weakness of right face, arm, and leg; talking nonsense. August 14: Seen by me; profoundly apraxic, no aphasia but speech apraxic; apraxia but no paralysis of right arm and leg; knee-jerks diminished, plantar reflex flexor, disks normal. Recovered entirely under inunction, followed by injections of mercury.

I need not delay you with describing the paralyses of cranial nerves, especially the various forms of ophthalmoplegia, which are of such frequent occurrence. Double vision frequently appears amongst the early symptoms, although the final manifestations may consist in a hemiplegia.

SPINAL CORD AND ROOTS.

Perhaps the commonest manifestation of spinal syphilis is spastic paresis with or without ataxy. Paraplegia, with a girdle sensation, is not uncommon, and in these cases again we find that the difference in the rapidity of the onset makes a material difference to the prognosis. Where the onset is sudden the prognosis is bad. Thus in Case X, nine months after infection, the patient suddenly lost power in the right leg, and the next morning found that the left leg was also paralysed completely. He was a typical instance of paraplegia without spasticity and with considerable loss of sensation. Two and a half years later he was still bed-ridden and had not altered materially.

Case X.—In January, 1907, at the age of 25, he developed a chancre, followed by a rash all over the body and constant sore throat. Treated for one month with mercury by the mouth in pills.

October 18, 1907: Sudden numbness over right half of body from the armpit downwards and over right leg; two hours later sudden paralysis of right leg; he woke next morning to find his left leg was paralysed and that he could not pass his water. Admitted to hospital on October 19, 1907: Absolute paralysis of motion below the pelvis; loss of sensation to pain, heat, and cold up to above the knees; right knee-jerk abolished, left just obtained with reinforcement; plantars not present, no ankle clonus; reflex incontinence of urine. At this time he showed a squamous and papular syphilide. In spite of treatment he did not recover, and still (1910) remains paralysed.

Contrast this with the sailor (Case XI) who, two years after infection, developed pain in the chest, which cleared up entirely. Five months later, during a similar attack, his legs gradually became weak, and he lost control of his sphincters. This man could not walk and showed all the signs of spastic paraplegia, but is now able to do his work well.

Case XI.—In 1903, at the age of 20, he was attacked with a discharge from the penis, which lasted more than four months and was followed by swelling of

the glands. On admission to hospital in 1906 there was a scar on the penis. He was treated at a general hospital.

March, 1905: Pains in the chest, especially at night, which disappeared entirely. August 18, 1906: A similar attack, which lasted until three weeks later; his legs became weak, and he had difficulty in holding his water. On attending the hospital in October, 1906, he could not walk without crutches; both legs were spastic and paretic and Romberg's sign was well developed; the knee-jerks were exaggerated, the left more so than the right; ankle clonus and Babinski's sign were present on both sides; pupils and disks normal; sphincter trouble had passed away.

Occasionally the disease manifests itself in a Brown-Séquard paralysis (Cases XII and XIII). In one case the left leg was spastic and paretic, and sensibility to pain, heat, and cold was lost over a great part of the right leg. But, unlike the cases of injury or hæmorrhage into the cord, these paralysees due to syphilis never conform strictly to the Brown-Séquard type. Thus in Cases XII and XIII, although one leg was affected with motor and the other with sensory paralysis, ankle clonus and Babinski's sign were obtained from both legs.

Case XII.—In October, 1905, at the age of 23, he developed a chancre, followed by frequent sore throats; six months later a rash of "small red pimples" was treated by a chemist for seven months.

February 15, 1908: Could not pass water; at night found he could not stand. March 9, 1908: Admitted to hospital; walked feebly, but could stand with feet together; left leg was spastic, and all movements below the knee were feebly performed; movements of the right leg were good, but the leg was a little stiff; definite loss of sensibility to prick, heat, and cold over the right lower extremity, occupying the sacral areas and the whole of the front of the leg below the knee; tactile sensation unaffected; girdle sensation at level of dorsal II; knee-jerks much exaggerated; ankle clonus from left foot occasionally; Babinski's sign from both right and left feet; could not pass urine. A macular syphilide was visible on the left thigh on admission. After six weeks' inunction he was enormously improved, and returned to work somewhat later.

*Case XIII.*¹—In June, 1902, at the age of 26, he developed a chancre, followed by sore throats and a rash. He was treated with mercury by the mouth for nine months.

In November, 1903, he was attacked with shivering pains in the back and loss of power in the legs, with retention of urine and incontinence of fæces. When admitted to hospital two weeks later there was loss of power in both legs, especially the left, and knee-jerks were absent; sensation to pain, heat, and cold was lost over the right leg, but normal elsewhere. He was

¹ The opportunity of seeing the case and embodying it in this report I owe to the kindness of Dr. Bertrand Dawson.

treated with iodide and with inunctions of mercury, and improved greatly. In June, 1905, when I saw him, the left leg was spastic and showed much loss of power; the right leg was much less spastic and the power was good; sensation to pain, heat, and cold was lost over the whole of the right leg, from the umbilical level downwards (fig. 1); tactile sensibility was everywhere perfect; the knee-jerks were exaggerated, the left more so than the right; both plantar reflexes gave an extensor response. He still had some difficulty in starting micturition, but the motions were passed normally.

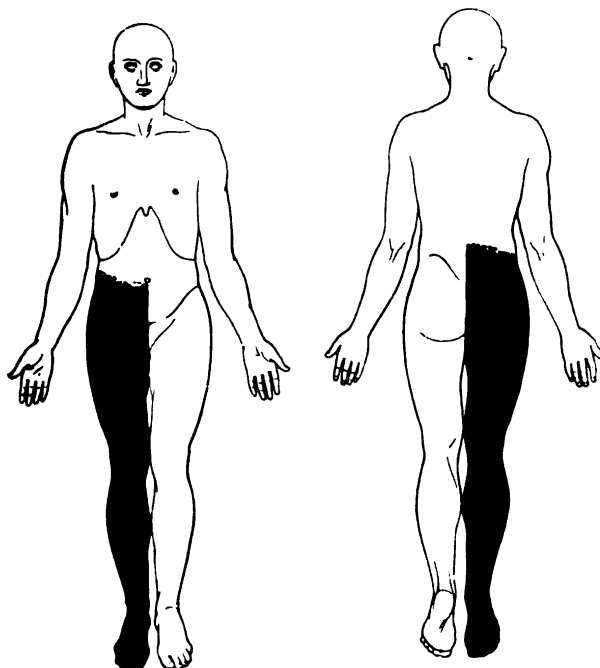


FIG. 1.

Root-lesions are a frequent concomitant of spinal syphilis. In one instance (Case XIV) of spasticity of both legs, without sensory changes or sphincter trouble, a superb root-analgesia extended from the back to the front in the region of the eighth and ninth dorsal. I believe such root-lesions are more often present than is usually supposed in spinal syphilis.

Case XIV.—In 1903, at the age of 28, his wife infected him with syphilis. Two years later sores came, without injury, on the left shin and ankle; the scars have the appearance usually produced by syphilis.

In 1906 he began to suffer from a numb feeling in the left side, as if something were gripping him, and found difficulty in walking; steadily grew worse for eighteen months, and since then has remained stationary. In July, 1909,

both legs were spastic and paretic, the left more so than the right. No wasting ; no ataxy ; no Romberg. Knee-jerks brisk and equal ; ankle clonus and Babinski's sign obtained on both sides ; sphincters unaffected. The only loss of sensation consisted of an area extending half round the body, as in fig. 2. Over this area touches with cotton wool were appreciated, but sensibility to tactile hairs (von Frey) was considerably lowered. Here also a prick was rarely recognized, and sensibility to pain was in many places lost. He was unable to recognize ice and water at 55° C., confusing the two stimuli, but could discriminate 22° C.

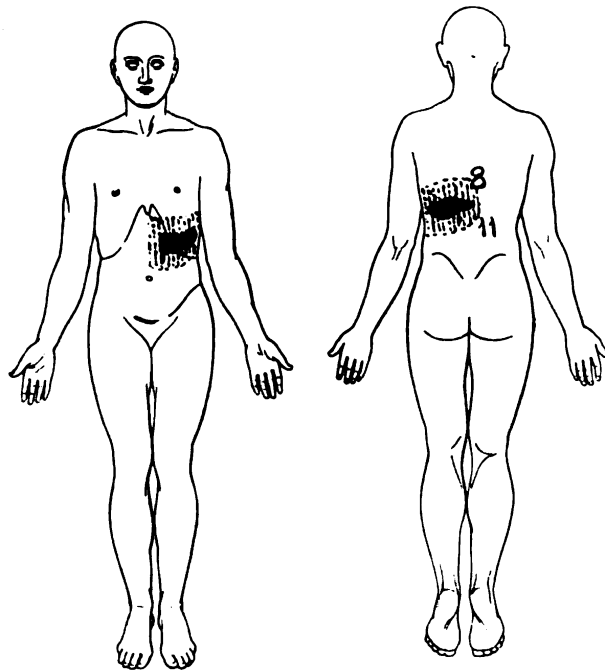


FIG. 2.

and 40° C. ; at the same time, 40° was said to be warmer than 50° C., and 22° C. colder than ice. This is a characteristic condition in lesions of the posterior roots. Treatment with inunction and rest, with, later, a course of iodide, improved his walking somewhat, but the root-lesion remained unaffected.

Disseminated syphilitic lesions are common in the early stages, but no careful observer would mistake such a case for one of disseminated sclerosis on account of the severity of the pains and headache, which do not usually accompany that disease.

In one case this condition came on seven months after infection. The speech became slow and slurred ; the right arm was tremulous and weak, both legs were spastic, and the patient was unable to hold his

water for any length of time. Here there was no nystagmus, but in another case, where the speech was unaffected and there was no tremor, nystagmus was present.

Case XV.—In March, 1903, at the age of 24, he developed a chancre, followed by a rash. He was treated for one month only.

In November, 1903, he developed pains in the legs and back, with weakness of the legs and incontinence of urine and fæces. In December, 1903, the right arm became numb, and three weeks later was paralysed. In November, 1904, his speech became slow and slurred. When seen by me in 1905 his speech was slurred. He had much occipital headache and pains in the neck. Right arm and both legs were spastic, and there was a coarse intention tremor of the arm. The knee-jerks were greatly increased, and ankle clonus and Babinski's sign were present on both sides. He was unable to hold his water for long. There was no nystagmus, and the disks were unaffected.

CEREBROSPINAL MANIFESTATIONS.

As we should expect from the pathology of syphilis, its nervous manifestations in the same case frequently point to lesions both of the brain and spinal cord. Thus one of my patients (*Case XVI*), a sailor, not only showed left ophthalmoplegia and right hemiplegia, but, in addition, definite sensory changes pointing to a lesion of the upper lumbar roots. Hemiplegia and paraplegia are a not uncommon combination, and many patients with so-called transverse myelitis will be found to have suffered from a lesion of one or more cranial nerves.

Case XVI.—In 1901, at the age of 26, he developed a chancre, followed by a rash.

In December, 1904, he had pain behind the right ear, followed by a complete facial paralysis fourteen days later. In May, 1905, he suddenly became drowsy, went to bed, and next day woke up paralysed in the right arm and leg. In August, 1905, he was admitted to hospital, walking on a wide base, with a spastic gait; he showed a true right hemiparesis, with typical right-foot drop and weakness of right hand; both knee-jerks were exaggerated, and ankle clonus and Babinski's sign were obtained from both sides; the left pupil did not react to light, there was ptosis of the left lid, and paralysis of the left superior rectus; at the same time he showed signs of a root-lesion in the left lumbar region and over the anterior portion of the right thigh. Thus this man had: (1) Right facial paralysis; (2) left ptosis and partial ophthalmoplegia; (3) right hemiplegia; (4) spastic paraplegia; (5) two distinct root-lesions.

CONCOMITANT SYPHILITIC MANIFESTATIONS.

In two out of thirty-three cases a secondary syphilitic rash was present at the time when the nervous affection appeared. One was a case of Brown-Séquard paralysis, the other showed the signs of transverse myelitis. A third case, two years after infection, suffered from a rupial eruption during the onset of a paraplegia. Finally, the case of apraxia (Case IX) suffered from a fresh attack of adenitis shortly before the onset of the cerebral symptoms.

TREATMENT.

The majority of cases yield to treatment with mercury, especially when applied by inunction after a hot bath. I have seen four cases where the patient was taking mercury by the mouth in the form of pills, under the guidance of an expert, at the time the nervous manifestations appeared. In one further instance spinal symptoms developed at Aachen whilst the patient was undergoing a full course of treatment, but was allowed to go about. All patients with nervous syphilis should, if possible, be put to bed, overfed, and massaged at the same time as they are treated with mercury by inunction. For in most cases the attempt to get about, or to perform work of any kind, adds to the strain on the affected nervous system.

DISCUSSION.

Dr. GORDON HOLMES showed a series of slides, in explanation of which he said that the point with which he would deal was that form which was spoken of as spinal syphilis, or syphilitic spinal meningitis, or meningo-myelitis. He had had the opportunity of performing post-mortem examinations on a considerable number of such cases, which was rare, though the condition was not infrequently diagnosed clinically. The main feature of the cases which he had seen was a thickening of the soft membranes of the spinal cord, which usually produced certain lesions in the cord itself. To only one of those cases, perhaps, could the term "meningo-myelitis" be applied, as in none of the others did the disease directly invade the spinal cord to a significant degree, the cord only suffering by compression and interference with its blood-supply. There were three points to which he would refer and illustrate by slides. The first was that the chief pathological lesion in the cord was a degeneration of the peripheral tracts, and especially in the longer tracts. The three cases shown

represented a fair average, and it was seen that the parts which had suffered most were the spino-cerebellar tracts, the direct cerebellar tract, and Gowers' tract. He correlated this with his experience that a considerable number of these cases of spastic paraplegia which result from syphilis present considerable ataxia, which has not the characters of that form due to a lesion of the posterior columns. The second point which the sections showed was that the dorsal columns escaped surprisingly well. He would rather say that, considering the amount of degeneration around the periphery of the ventro-lateral columns, there was remarkably little degeneration of the dorsal columns; and that was more surprising, in regard to the fact that Dr. Mott had emphasized that the dorsal columns were those which suffered predominantly in parasymphilitic conditions; and, secondly, because many authorities, as Nageotte, Obersteiner, and Redlich, have attributed the degeneration of the roots and of the dorsal columns in tabes to meningeal disease. The third point had already been brought out by Dr. Head—namely, the remarkable diffuseness and multiplicity of the lesions in the cases where one could examine the whole central nervous system. He would like to hear the opinions of others on the subject, but in almost every case in which he had seen a cerebral gumma where the full examination of the brain had been possible, there had been more than one gumma. Secondly, there was rarely a localized meningitis of the spinal cord, and therefore he thought the value of local treatment might be over-estimated. Further, not only was the meningeal disease not limited to any region of the spinal cord, but it generally extended over the whole central nervous system. The third thing which his cases illustrated was that frequently the meningeal disease was associated with softenings, multiple or single, throughout the central nervous system.

Dr. WILFRED HARRIS said he thought the Section should feel much indebted to Dr. Mott, Dr. Head, and Dr. Gordon Holmes for their contributions. Dr. Mott raised one very important point from the point of view of diagnosis, and he would be glad to hear from him an explanation on a point which he was not quite clear about. It was as to the reason—if one could be given—why, in those metasymphilitic conditions, general paralysis and tabes, there should be a positive reaction to the Wassermann test of the cerebrospinal fluid, whereas in more active and definite syphilitic disease of the nervous system, such as cerebrospinal syphilis, the Wassermann was negative in the cerebrospinal fluid. In active syphilis in other parts of the body one found a positive Wassermann in the serum. So long as there was a potential activity of syphilis in the system, so long, it seemed, there would be a positive Wassermann reaction. And one might expect to find it in the cerebrospinal fluid where there was active syphilis in the central nervous axis. But apparently it was not so. Yet in general paralysis and tabes—which later most observers agreed was not an active syphilitic process—the Wassermann reaction was positive in the cerebrospinal fluid as well as in the blood.

Dr. Head had referred to convulsions and epileptiform attacks in the early years following syphilis. That was a very important point, and one which was not sufficiently appreciated. In the last twelve months he had come across three cases of what he would call syphilitic epilepsy—one woman and two men—arising apparently without cause. And on hunting for a cause, as one should do in epilepsy, especially if there was no evidence of heredity, in each of them he found contracted pupils, which either did not react to light or did so very badly. That gave him the hint that the condition might be of syphilitic origin. But he could get no positive history of syphilis in any of them, though in each there was a very positive Wassermann reaction. What the exact cause of the epilepsy was he supposed one could not be certain, but the strong probability was that it was a diffuse meningitis, one or two irritative patches causing the attacks. Dr. Head also referred to Brown-Séquard paralysis and the clinical signs of the differentiation of sensation, and that it tended to be bilateral. One would expect that; for there would be nothing approaching a pure hemisection of the cord. One could not get such an anatomical lesion in such a diffuse condition as cerebrospinal syphilis; and it was curious that the symptoms of Brown-Séquard paralysis were so clearly marked as they sometimes were. He asked whether others had seen the same as he had, that crossed analgesia did not rise anything like as high as the motor symptoms on the opposite side. He remembered Dr. Head showing a picture of a case of fracture-dislocation of the spine in which that point was referred to. In that case he believed the left arm was more or less paralysed, and there was a level of root anæsthesia somewhere round the first dorsal on one side, whereas the crossed analgesia on the other side did not rise within some six segments of that. He had had a case of syphilis in the cord showing Brown-Séquard symptoms something like those in rheumatism, severe pain in the neck and down the arm for weeks; the patient then developed severe wasting and paralysis of his left arm, and weakness of his left leg. On examination there was found to be root wasting of the left arm, spastic paralysis of the left leg, but hardly any weakness of his right leg, though certainly increased knee-jerks, total analgesia of the right leg up to the groin, but no higher, the symptoms suggesting that there was a gummatous lesion in the region of the fifth and sixth cervical segments on the left side, and yet the crossed analgesia on the opposite side was no higher than the groin. He had seen similar symptoms in three cases in which the analgesia rose no higher than the groin, although the gumma was probably much higher up. One point on which he had hoped to hear more was the primary tract degenerations in the spinal cord, such as had been described as Erb's syphilitic spinal paralysis, combined sclerosis, etc. He (Dr. Harris) took it that most of the cases of sclerosis arose in the way Dr. Gordon Holmes had demonstrated—viz., due to lesions of vessels from a thickening of the lepto-meninges cutting off the blood-supply to the peripheral white matter, and so causing ascending and descending degeneration. But there were records of carefully worked out cases in which there seemed to be primary pyramidal degeneration;

and the question was if they were invariably due to some higher lesion, such as Dr. Holmes showed in the pons; or were they cases of post-toxic primary degeneration? They could not be due to lesions of vessels, to any endarteritis obliterans; they must be either post-toxic or due to some symmetrical lesion higher up. Various cases have been published under the name "Primary tract degenerations." If they were truly primary, that must bring them into line with the metasymphilitic disease—namely, tabes. Dr. Head also referred to hyperplastic infiltration of syphilitic new growths, but he did not refer to primary degenerations, under which he (Dr. Harris) would include cell-degenerations. The evidence proved that there was such a thing as a condition starting subacutely and becoming chronic poliomyelitis. He had seen three such cases, and he thought they might be classed with the cell-degenerations which were met with in general paralysis of the insane.

Dr. GRAINGER STEWART remarked that there was not much to be added to what had already been said. He thought more attention must be devoted to the point made by Dr. Head as to the relationship of epilepsy and syphilis. He had recently come across two cases in which there was no family history of epilepsy or personal history of convulsions in infancy. One was a girl aged 18, and the other a woman aged 23. In the elder patient the fits started when she was aged 15. He obtained conclusive evidence that she had been infected with syphilis, and had been treated for this disease a year before the fits began. He put her upon antisymphilitic treatment and she had greatly improved. With regard to the diagnosis between disseminated sclerosis and disseminated syphilis, he thought that in most cases it was obvious if the history of the case was known and one had been able to watch it. Still, there were many cases in which young people who had had syphilis exhibited symptoms which at first might be attributable to either disseminated sclerosis or to early manifestations of syphilitic lesions. In these cases one was obliged to resort to the examination of the cerebrospinal fluid, or to the Wassermann reaction, because of the importance of giving the patients the benefit of being properly treated from the syphilitic point of view. With regard to Dr. Holmes's remark concerning the treatment of local lesions in syphilis, the point to consider in regard to whether local interference should be undertaken was whether the lesion was causing destruction of the nerve tissues. If there was evidence that it was occurring, one was compelled to advise operation. Dr. Mott had already referred to a case of pachymeningitis in which he thought the patient would have been improved by operation. In cases of paraplegia due to syphilitic spinal meningitis, he thought the presence of root-lesions was a great help in diagnosing the level; and in such cases, provided that antisymphilitic treatment had not done good, one should not wait, but should give the patient the chance of benefiting by an operation undertaken with the view of relieving the pressure upon the cord. Certainly in his experience, in some cases, antisymphilitic treatment had been more beneficial after operation.

Dr. STANLEY BARNES regretted that more stress had not been laid upon the conditions under which muscles atrophied in post-syphilitic conditions. He had come across many cases in which it was uncertain whether one was treating a syphilitic or a parasyphilitic condition. He had one patient in mind who was under his treatment six months—a young man who within two and a half years before he saw him had acquired syphilis. He had had intense mercurialization, and had had 100 gr. of iodide three times a day, but with no effect in checking the progress of a flaccid paralysis in one arm. It increased so rapidly that seven months from the starting of the weakness he was almost entirely paralysed in the left arm, and the flaccid paralysis was commencing in the other arm. Was that condition a parasyphilitic or a primary syphilitic one? Was it a degeneration of the nerve cells, or was there a coarser lesion which accounted for the condition? If, as he presumed, it was parasyphilitic, it was very unusual for it to occur at so early a stage after the syphilis had been acquired. The diagnosis from disseminated sclerosis was sometimes difficult, as in the latter disease root-pain was occasionally a prominent symptom. He had been surprised not to hear more of syphilitic epilepsy, but he was glad to have heard Dr. Grainger Stewart and other speakers refer to it. He had seen eight such cases in the last twelve months, epilepsy occurring in an individual who had had syphilis, but in whom no other ætiological factor than syphilis could be found. And yet he had been sadly disappointed in the results of treatment in those cases. That might be due to the presence of adhesions which had already formed between the meninges and the cortex, causing a permanently irritable focus in the brain. In one such case Jacksonian attacks were present. He had had the gumma excised, but the case still required bromide to keep the attacks under. The attacks while under bromide treatment were only sensory ones, but whenever treatment was relaxed definite Jacksonian attacks still occurred, although the gumma was excised two years ago.

Neurological Section.

February 24, 1910.

Professor C. S. SHERRINGTON, F.R.S., President of the Section,
in the Chair.

Tumours of the Pineal Body.

By C. M. HINDS HOWELL, M.D.

TUMOURS of the pineal body are of considerable interest both from a clinical and pathological point of view. In addition, evidence is slowly accumulating which seems to indicate that this structure, although only a rudimentary and degenerating organ in man, may exercise a peculiar influence on development and metabolism, at any rate in the first few years of life. No doubt the rarity with which tumours have been found associated with the pineal body accounts in part for our lack of more definite information on this subject. Comparatively few cases are recorded in the literature of the subject in this country, though abroad, and especially in Germany, more cases have been reported. Within the last year two complete cases have occurred at the National Hospital, and I have been able to find one other in the hospital records. I have to thank Dr. Ferrier, Dr. Risien Russell, and Dr. Collier for kindly allowing me to refer to these cases here to-night.

Case I.—P. L., a man aged 42, was admitted to the National Hospital under the care of Dr. Ferrier in 1905. The following is a brief extract of his notes, which were taken by Dr. S. A. K. Wilson :—

Family history and previous history good. Present illness : For six months before admission the patient had complained of dimness of vision and occipital headache ; the latter was severe for four months and then had become much less. In addition, the patient complained of giddiness with the sensation of being forced towards his left side, and also of a tendency to fall backwards. When walking he staggered to the left. There had been diplopia for one month before admission.

Examination of the central nervous system: Smell and taste were normal; recent optic neuritis; no ptosis, but upward movement of the eyes was lost, while downward movement was much impaired, especially in the right eye; lateral movements were good; the pupils were small and inactive to light and convergence. Motor system: No paralysis. Sensory system: Normal; reflexes unaffected, save that the left abdominal reflex was diminished. Operation was performed over the cerebellum for the relief of pressure. The patient died suddenly, six weeks after the second stage had been performed.

The post-mortem examination was made by Dr. Gordon Holmes. The convolutions of the brain were considerably flattened and the infundibulum distended with cerebrospinal fluid. The pons was widened and flattened out, as were also the peduncles and ventral portion of the mid-brain. A swelling was seen in the dorsal region of the mid-brain, and, after hardening, a tumour was found extending from the posterior commissure, where there was a dorsal projection of growth to the valve of Vieussens. The medulla, pons, and cerebellum were free from growth. No special note is made of the pineal gland. Sections cut through the posterior corpora quadrigemina showed the tumour occupying and distending the Sylvian aqueduct and the dorsal part of the mid-brain. It seemed to be distending the tissues rather than infiltrating. The structures ventral to decussation of superior cerebellar peduncles were normal. Section through the anterior corpora quadrigemina showed the tumour increased in size, and still displacing rather than infiltrating the tissues. A projection had taken place ventralwards separating the red nuclei. The tumour ended by slightly infiltrating the mesial parts of the thalami on either side, but did not extend so far forwards as the soft commissure. Examination by the Marchi method showed a diffuse degeneration, but definite tract degeneration could be distinguished in the rubrospinal tract (slight), the central tegmental tract of right side, and the right tectospinal tract.

The microscopical examination of the tumour I shall describe later, as in all three of these cases the growths presented very similar features.

Case II.—B., a boy aged 22, admitted to the National Hospital under Dr. Collier, February, 1909.

Previous health was excellent. Present illness: Has complained of nausea with slight sickness for some months. Three months ago had to stop work owing to vertigo, and at this time was told that his eyelids drooped. For six weeks has been getting stupid and inattentive, and has noticed tremor in his hands and uncertainty in his gait. For two weeks has complained of headache and diplopia. On admission was found to be a healthy-looking boy; exceedingly restless. He was somewhat deaf, though this was very slight; complained exceedingly of thirst and drank enormously of water. Nervous system: There was marked optic neuritis, the pupils were equal, slightly ectopic (upwards and inwards), and inactive to light and accommodation; there was complete loss of upward movements of the eyes, the effort producing side-to-side oscillations; downward movement was very incomplete, and there was slight

paresis of the left external rectus, lateral movements being otherwise well performed; there was slight ptosis; the other cranial nerves, with the exception of the eighth, which was slightly affected, were normal. Motor system: The power of the limbs was good; a rhythmical tremor was present



FIG. 1.

Section through temporo-sphenoidal lobes and mid-brain in Case II, showing tumour distending the third ventricle.

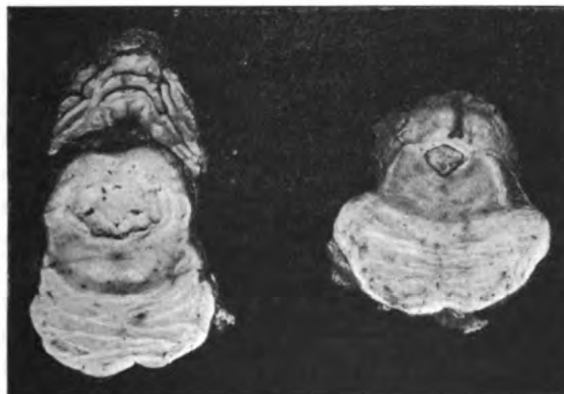


FIG. 2.

Sections through pons in Case III, showing tumour within the Sylvian aqueduct.

in both upper extremities, which affected the hands most; ataxy on both sides; lower limbs normal. Sensory system: Apparently normal; gait very uncertain, without any particular ataxy, "the knees seem to give way." Reflexes

call for no comment. A curious feature was the way in which the boy's upper limbs shuddered periodically, as though he were very cold. This occurred without any subjective feeling of cold being experienced in the least.

I made the post-mortem examination in this and the following case. The convolutions of the brain were very much flattened and the floor of the third ventricle was bulged out as a thin-walled cyst under considerable pressure. On raising the cerebral hemispheres the dorsal part of the mid-brain appeared enlarged and bulged out, whereas the ventral part was decidedly flattened. Horizontal sections made through the hemispheres showed considerable hydrocephalic dilatation of the ventricles. A growth was found projecting into the dilated third ventricle from behind. In size it resembled a walnut and was somewhat friable in consistency. It had not infiltrated the thalami, but seemed rather adherent to the ependyma of the ventricle. The tumour was covered by velum interposition, which could, however, be easily stripped from off its surface. The posterior commissure had apparently been destroyed, and no trace of the pineal body apart from the growth could be found. The Sylvian aqueduct was much dilated and filled with growth, which had eventually distended the canal so much that it had burst through the roof. A process of growth had continued down the aqueduct, which it completely filled, into the commencement of the fourth ventricle. It had not infiltrated the walls of the aqueduct to any appreciable extent, nor involved the third nerve nuclei or red nuclei directly. There was no hæmorrhage or softening in any part of the brain-stem. The spinal cord was normal.

Case III.—Admitted to the National Hospital under the care of Dr. Risien Russell on October 4, 1909.

F. L., a boy aged 20. Previous history excellent, except for attacks of malaria. Present illness: Commenced five months before admission with constant frontal headache and general weakness following a malarial attack. The legs were said to give way when attempts were made to walk. He complained of failing memory and a sensation of giddiness, which was not accompanied by any definite sense of rotation. Examination of nervous system: Smell somewhat defective; intense optic neuritis, pupils central, very sluggish reaction to light and convergence, tendency to assume an oval shape on dilatation, complete loss of upward movements of eyes, nystagmus on lateral deviation; slight left facial weakness; hearing acute; no other cranial nerve symptoms. Motor system: Considerable weakness in left arm and leg and great weakness of back muscles, marked tendency to fall over backwards when sitting up; some inco-ordination, right arm; no tremor. Sensory system: Normal; reflexes all normal and brisk. The patient died after an operation in the posterior fossa, designed to relieve intracranial tension.

On removal of the brain the convolutions were found to be extremely flattened, and there was great hydrocephalic dilatation of the ventricles, evidenced by the distended floor of the third ventricle and infundibulum. The pons and ventral aspect of the mid-brain presented the same flattened appearance

which has been noticed in the two previous cases. As a result of the distension of the third ventricle, the optic chiasma had been considerably compressed. The brain was divided by coronal sections after dividing the brain-stem. An extreme degree of hydrocephalus was found to be present, caused by a tumour, apparently of the pineal body, which had grown forward into the posterior part of the third ventricle and blocked the Sylvian aqueduct. As in the last case, the growth had been forced down in this, distending it, and causing a rupture through the upper part of the roof. There had been slight infiltration of the mesial aspect of both thalami, and the growth had been limited above by coming into contact with the splenium of the corpus callosum, which, however, it had not infiltrated except very superficially. No trace of the pineal body apart from the tumour could be found. Sections were made through the brain-stem, pons, medulla, and cord (first cervical segment), and stained by the Marchi method. Very well marked degeneration was present in the dorsal longitudinal bundle throughout all sections, and in the ventral longitudinal bundle so far caudalwards as the lower end of the pons. There was a fine diffuse degeneration in the central tegmental tract of either side and in both superior cerebellar peduncles. There was also some coarse degeneration in the fibres of the mesial fillet throughout the pons; the rubrospinal tract showed no degeneration.

With regard to the character of the tumours present in these three cases, there could be no doubt that all were essentially the same. The tumour proper was composed of rather large cells with round or oval nuclei, surrounded by an ill-defined zone of protoplasm which stains in a very indifferent manner with acid picro-fuchsin. In the central part of the tumour these cells are arranged in masses, with very little intercellular substance. Towards the periphery, however, the cells have a definite alveolar arrangement, masses of from eight to twenty lying within compartments the walls of which are formed of thin fibrous tissue. In places in the tumours, especially in the first and second case, and also, though to a less degree, in the third, the fibrous septa are very pronounced, and along them were columns of small round cells which closely resemble lymphocytes in appearance, though in places plasma cells may be detected among them. These columns of small cells follow the lines of the fibrous septa most closely, and it is further to be noted that the blood-vessels in the growth also follow the same lines. In the third case there is evidence of a good deal of chronic inflammatory tissue within the tumour, and here and there giant cells of inflammatory origin may be seen. The larger blood-vessels, both within the tumour and in the brain tissue immediately adjoining, show very intense perivascular small cell infiltration. In none of the cases were the ocular nuclei, or red nucleus, involved by the growth, so that the

palsies noted in all the cases, and the tremor noted in the second case, must have been due only secondarily to the growth.

As to the exact nature of the tumours it is hard to decide. In some respects they resemble the structure of the normal pineal gland. In this I would remind you there is a definite alveolar arrangement, the cells of the alveoli being formed by fibrous tissue, along which run the blood-vessels. Within the alveoli the cells of the pineal body may be seen. These consist of a well-staining nucleus surrounded by ill-defined protoplasm, and are often stellate, especially towards the periphery of the alveolus, which has no definite endothelial or epithelial lining. There is a large amount of intercellular substance. I was unable to



FIG. 3.

Microphotograph of normal pineal body, showing alveolar arrangement.
(Low power.)

find any evidence of pigment in any of the cells or of any nerve fibres within the pineal body. The chief tumour cells resemble those of the pineal very closely, but are definitely larger, and never exhibit any of the stellate forms seen in the gland. Personally, I do not think the columns of small round cells are primarily a part of the tumour, but in my opinion they are inflammatory in origin. They are best seen along the thicker strands of fibrous tissue.

It is interesting to note that Carl Hart, under the title "Angiosarcoma of the Pineal," describes a tumour which in many respects resembles those met with in these three cases, and the tumour described by Lawrence has some points in common also. Duffin and Ferrier reported a case in the *Transactions of the Clinical Society*

which, in its distribution within the third ventricle and Sylvian aqueduct, closely resembles these three cases I have reported to-night. A further point of resemblance was that it had caused compression with atrophy rather than infiltration of the tissues. The clinical symptoms also were very similar, though the ocular palsy was confined to slight weakness of the external rectus on the right side. The most intense giddiness, relieved somewhat by closure of the eyes, was a prominent symptom, as was also early failure of vision and deafness. The tumour in this case was described as a glioma. Whatever nomenclature we adopt for these

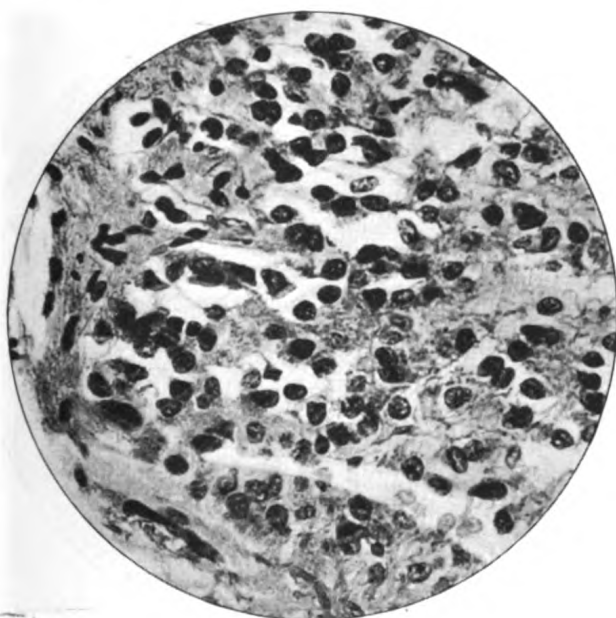


FIG. 4.

Microphotograph of normal pineal body, showing nuclei surrounded by the defined protoplasm. (High power.)

tumours, it is evident that they possess only a low grade of malignancy, as is shown by the fact that they have little tendency to infiltrate adjacent structures, and apparently give rise to no metastatic deposits.

Certain interesting points arise in connexion with the symptomatology of these cases. The patients, as a rule, are males, and the age-incidence seems to be in young or early adult life. In only three cases of those I have been able to find was the patient over 30 (C. Ogle, aged 32, melanotic sarcoma; Verger, aged 42, no histological description; and Dr. Ferrier's, the first case to-night, aged 42). General symptoms, such as headache,

vomiting, and optic neuritis, have been present in almost all the cases, and usually well marked. The headache is, as a rule, occipital, and frequently associated with pain and rigidity in the neck muscles; sometimes a certain degree of opisthotonos is associated with this. There are certain other general symptoms which would seem to have some importance as aids in diagnosis from the frequency with which they occur. The most striking of these is sleepiness, which was a marked feature in the cases of Ogle, Coats, Frankl-Hochwart, Gutzeit and Duffin. In addition, languor and an apathetic mental condition are

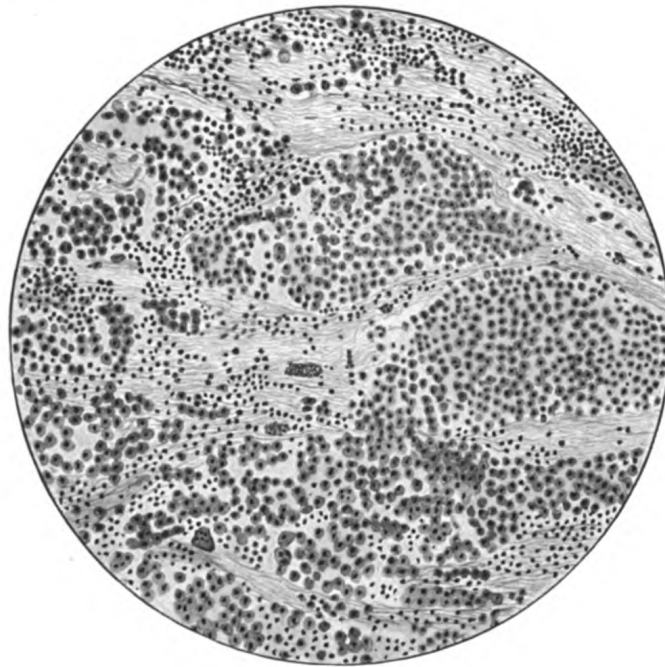


FIG. 5.

Drawing of section of tumour in Case II. ($\frac{1}{4}$ in. objective.) Note alveolar arrangement and small cells along fibrous tissue.

frequently mentioned. Two curious symptoms were present in the second case in this paper, and in a case recorded by Daly. In the first the patient had an insatiable thirst, whilst in the second the appetite was enormous, the patient gaining 5 st. in weight during the comparatively short time he was under observation before his death. Owing to the position of the pineal gland, tumours arising in connexion with it may be expected to give rise to symptoms typical of a lesion involving the mid-brain and corpora quadrigemina. This they usually do, though

a not inconsiderable number have given rise to ambiguous symptoms, resulting usually in the diagnosis of cerebellar tumour, more rarely of tumour of the pituitary body. The characteristic symptoms may be grouped in order of their localizing value into ocular palsies, vertigo, staggering gait, tremors and deafness, to which may be added the pain and rigidity in the neck sometimes associated with definite opisthotonos, to which reference has already been made. Examining these symptoms a little more in detail, one finds the most constant of the ocular disturbances to be affection of the pupils. These, as a rule, are dilated, and

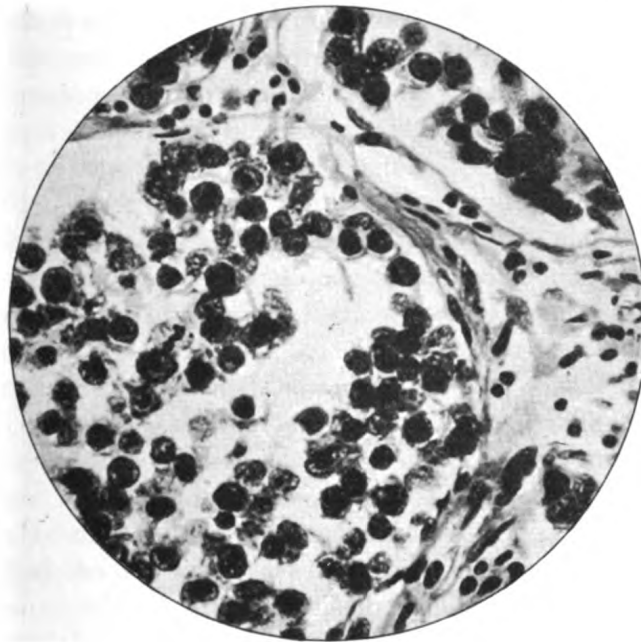


FIG. 6.

Microphotograph (high power) of tumour in Case III, showing alveolar arrangement and cells with ill-defined protoplasm round nuclei.

very early either quite inactive or very sluggish to light; later they become inactive to both light and accommodation. Next in frequency paralysis of the external ocular muscles takes place, that met with earliest being loss of conjugate upward movement of the eyes, which is sometimes followed by loss of downward movement. As we have noted in the three cases reported to-night, the ocular motor nuclei were in no case directly invaded by the growth, and these results must be of a secondary character; the centre for upward movements lying furthest

forward would, in all probability, be soonest exposed to pressure, and their early paralysis would be thus explained. The loss of upward movement is, however, not a constant symptom, and cases are reported in which movement in this direction was free, whilst the external rectus on one or both sides was paralysed. In other cases, again, the external ocular muscles seem to have escaped altogether. An interesting fact which has been recorded in several of the cases, notably by Duffin and Ferrier, Ogle, and Daly, is the rapid loss of vision which has taken place. In Ogle's case this was unaccompanied by optic neuritis. This author offers the explanation of a lesion of the anterior corpora quadrigemina to account for the visual defect, but accumulated evidence makes it probable that these nuclei are not directly connected with vision, and some other reason must be sought. Perhaps the internal hydrocephalus, which in many instances is very great, may assist in producing loss of function by direct pressure on the chiasma.

The remaining symptoms do not call for any very special comment. The vertigo, which may be most intense, is usually described as a general giddiness without any definite sense of rotation, subjective or objective. The gait is sometimes of the cerebellar type, and in others is described by the patients as being due to their "knees giving way," and this, too, without the presence of any real motor weakness. The tremors present are often of the type associated with disseminated sclerosis, and may be due to involvement of the red nucleus or its connexion either with the superior cerebellar peduncle or the spinal cord.

In this connexion we may note that Ferrier was able to produce tremors of this type by division of the superior cerebellar peduncle. The deafness which has been noticed in one or two instances may have been caused by pressure on the posterior corpora quadrigemina, but the clinical data are not recorded with sufficient accuracy to enable one to lay much stress on this point. The motor and sensory systems, as a rule, are unaffected.

The symptoms we have considered are those exhibited by adults, but a remarkable complex associated with pineal tumour has been described in the case of children. Cases exhibiting this complex have been described by Ogle, Frankl-Hochwart, Heubner, Gutzeit, Marburg, Oestreich and Slawyk. In all these cases except one the patient was a boy, and in no case was the age above 10. In addition to signs of intracranial tumour, and, as a rule, dating from the first appearance of symptoms, undue development, both psychical and physical, has taken place. The children have become wise beyond their years, and in some

cases apparently interested in philosophical discussions. The physical changes have been marked: excessive growth of the body generally, including bones and muscles, has taken place, and with this an altogether abnormal development of the external genital organs, with growth of pubic and axillary hair, and distinct indications, into the bargain, of moustache and beard, has been noted; the voice has become deeper. In Marburg's case, that of a girl aged $9\frac{1}{2}$, the external genital organs seem to have remained unchanged, but the child became pathologically adipose, the fatty tissue being chiefly noticeable in the breasts and abdomen.

It is difficult to say what the explanation of these remarkable changes may be. In formulating any theories one must remember that much the same symptom-complex has been noticed in association with suprarenal growths and pituitary tumours. In the latter case the genital change appears to be rather in the direction of atrophy than hypertrophy, but enormous fatty development takes place. Launois and Cleret have recently recorded twelve cases of this type associated with pituitary lesions. Further, Sacchi has described a case, analogous to these, which arose in connexion with a tumour of the testicle in a boy aged $9\frac{1}{2}$, and instances of undue fatty development have been recorded in connexion with tumours of the cerebellum, and by Nothnagel in connexion with tumour of the corpora quadrigemina. With the evidence at present at our disposal we must obviously hesitate to assign the symptom-complex under discussion to a lesion of the pineal body. Marburg, however, maintains that during the early years of life, at all events, it has a definite function, and that lesions may result in one of three consequences:—

(1) Premature genital development, which he regards as a hypopinealismus;

(2) Universal adiposity (hyperpinealismus);

(3) Cachexia (apinealismus).

At the same time he regards the pituitary as exciting an inverse form of influence: thus *hyperpituitarism* causes genital hypertrophy, and so on.

Askanazy, on the other hand, holds the view that these changes depend not so much on the pineal body as on the nature of the tumour by which it is involved. In the majority of recorded cases the growth was a teratoma. This Askanazy regards as "false pregnancy," a retrograde step towards the early views as to the pathology of these curious tumours when they were regarded as "offspring of the devil." Objections

to this assumption at once occur to one, as teratomata frequently exist without producing such symptoms, and in the case of the pineal body never seem to give rise to them unless developing during early life. From a physiological point of view the pineal body does not seem a very promising subject for the development of an internal secretion. In man, at all events, and in most of the vertebrates, it is a degenerate relic of the paired median eyes of the invertebrate, and its structure does not suggest any glandular activity. Further, Dixon and Halliburton have undertaken a research into any possible physiologically active principle that the gland might contain. They worked with sheep's pineals, which they extracted in different ways, and tested the extract by intravenous injections into anæsthetized cats. Only when large quantities of extract were injected (5 c.c.) was any effect obtained, and that, too, was only a small fall in blood-pressure (this result was proved not to be due to cholin). The authors conclude that their experiments throw no light on any possible function of the pineal body, but one must remember that the remarkable symptoms associated with these tumours only occur in very early life, and possibly the glands used may have become functionless.

With regard to the nature of the tumours associated with the pineal body, one is struck by the fact that so many of them are mixed forms, and not a few are teratomata. It is difficult to classify exactly the class of tumour which was present in the three cases I have described to-night, and the same difficulty occurs in many of the other cases. A noteworthy feature is the similarity of these tumour cells, in many instances, to those of the normal pineal body.

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DISCUSSION.

Sir VICTOR HORSLEY, F.R.S., said that he had been extremely interested in Dr. Howell's paper because he had seen the cases which Dr. Howell had described, and he had also seen another case some years ago at University College Hospital. The point that impressed him in all these cases was that the patients showed not only the ordinary symptom of loss of movement in the eye, but also the cerebellar attitude, due to the pressure on one of the peduncles. In each case, as it happened, it was the right cerebellar attitude that was noted. The case that he had seen at University College Hospital was under the care of Dr. Russell, and when first seen the patient presented to so great a degree the mental phenomena that Dr. Howell had described that it was thought to be a case of frontal tumour, which diagnosis had afterwards to be revised. It was quite obvious that the mental changes must be due simply to intraventricular distension, but the cerebellar attitude due to the pressure on the peduncle, and also pressure backwards to the right cerebellar region, was, of course, also confirmed by the tendency of the patients to fall backwards. Each of these cases showed this symptom in a marked manner. On the patient sitting up, even at the edge of the bed and in the most comfortable position, he fell backwards as soon as his spinal column became vertical. The situation of the tumour explained the remarkable constancy of the symptoms in all these cases. With regard to the side of the question in which he felt a special interest—namely, the possibility of doing anything surgically—he was bound to confess that the surgical results so far were far from favourable. He thought this might be due to the fact that in each case he had approached the lesion subtentorially. In the next case with which he had to deal he would certainly go supratentorially, splitting the tentorium from the ventro-posterior position and exposing the dura in that manner. He suggested that all future decompression operations, or all operations for the removal of these tumours, should be supratentorial instead of infratentorial. The Section was much indebted to Dr. Howell for gathering together this material and presenting it in so lucid a manner.

Dr. GORDON M. HOLMES said that Dr. Howell's contribution had interested him very much, for the reason that he had performed post-mortem examinations upon two cases of pineal tumour, one of which—the one under Dr. Ferrier's care—had already been referred to by Dr. Howell. When he examined that tumour histologically, he was entirely nonplussed; but about a year ago he had the opportunity of performing a post-mortem examination on another case, a boy of about 18 or 20 years of age, and in that case the tumour lay on the surface of the corpora quadrigemina. When he examined the tumour histologically he found it to be absolutely identical with that in Dr. Ferrier's case, and as the tumour in his second case was, without any question, in connexion with the pineal gland, he felt justified in interpreting the first in the same way. He was glad to find that his conclusion on the pineal origin of the tumours had been confirmed by Dr. Howell. With regard to the clinical symptoms, he had nothing to add to what had already been said by Sir Victor Horsley. But from the surgical point of view he thought that two things should be kept in mind. In the first place, such tumours might penetrate the third ventricle and diffuse through the ventricular system; and, on the other hand, they might lie detached on the corpora quadrigemina, and in such a case he thought a surgeon might be able to remove them. When they saw a case progressing from bad to worse they might very well hope to find such a localized tumour in that region, and to hand it over to the surgeon. Dr. Howell had mentioned tremor, as a distinctive feature of one of the cases he had described, but he happened to remember that in Dr. Ferrier's case also there was definite tremor before death. In that case, as Dr. Howell had said, there was degeneration of the rubrospinal tracts. Finally, he might mention a fact of special interest to the Section. One of the last cases that Dr. Beever brought before them was a tumour of the mid-brain; the man had since died, and, from a short note appended to the pathological report of the case which had been published by Dr. Judson Bury, it appeared that the tumour was similar in structure to those to which Dr. Howell had referred that evening, and which he (Dr. Holmes) had examined.

Dr. HINDS HOWELL, in reply, said that with regard to the nature of the tumour reported by Dr. Judson Bury, he did not think that this was similar to those which had formed the substance of his paper. The dissimilarity was found in the entire absence of fibrous tissue within the tumour in that special case, whereas in the tumours that he (Dr. Howell) had examined the fibrous tissue within the tumour was the most striking feature. Dr. Bury's tumour was also an infiltrating one, and no mention was made of any connexion with the pineal body in his paper. For these reasons he (Dr. Howell) did not feel inclined to include it in his paper as a pineal tumour.

A Case of Pontine Thrombosis causing Anæsthesia of the Fifth Nerve and Hemianæsthesia of the same side.

By H. CAMPBELL THOMSON, M.D.

This patient, a man aged 64, stated that he went to bed as usual on December 27 last, and woke up at 2 a.m. feeling weak all over: that he rose in the morning but felt, in his own words, that his circulation had stopped in the left leg, and that he could neither stand nor walk down stairs. He got the leg massaged, and improvement quickly took place so that he could get about. He also complained of numbness down the whole of the left side of the body.

I did not see him until January 8, 1910—that is, nearly a fortnight after the attack. There was then no sign of damage to the motor fibres, there was no loss of power, his knee-jerks were equal and normal, and the plantar reflexes were both of the flexor type. There was, however, ataxy in the left leg and he always inclined to the left, and every now and then gave a lurch in that direction. He had lateral nystagmus on turning the eyes to the left; none on the right. There was no paralysis of any ocular muscle.

There was complete anæsthesia over the left half of the face. He could not feel touch, nor could he recognize pain or temperature sensations. Over the trunk there was definite diminution of sense of temperature, and some slight diminution of sensations of touch and pain. Over the left leg the temperature sensation was not quite normally acute, but there appeared no alteration to the other forms of sensation and scarcely any alteration at all could be detected in the arm (fig. 1). He had for some time been rather deaf on both sides, but volunteered the statement that he was more so on the left since his illness, and, as he was an intelligent man, I felt able to accept his statement as probably true. The sense of taste was lost on the left side of the tongue, the palate moved freely, there was no laryngeal paralysis, no paralysis of the left facial and no hemianopia. The sense of smell was not affected and there was no weakness of the motor side of the fifth nerve. From what he said he was obviously already a good deal better when I saw him, so it is probable that the sensory changes were more marked at the beginning of his illness.

He continued to improve, and on February 19, when I last examined him, there was little alteration to be noticed in his walking. His nystagmus, on turning the eyes to the left, still persisted, and his sensory condition was as follows: On the left side of the face over the area corresponding to the fifth nerve he could feel light touch quite well; but the sensation of pain was entirely lost, as also was that of temperature to a great degree, though he could now just distinguish between extremes of heat and cold. Over the rest of the body sensation had almost entirely returned, the only things noticeable being a slight failure in the appreciation of extremes of heat on the left side of the chest and abdomen.

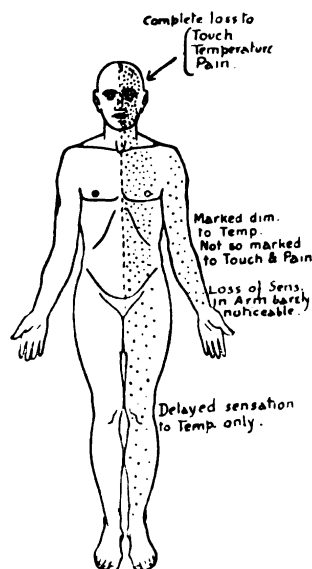


FIG. 1.

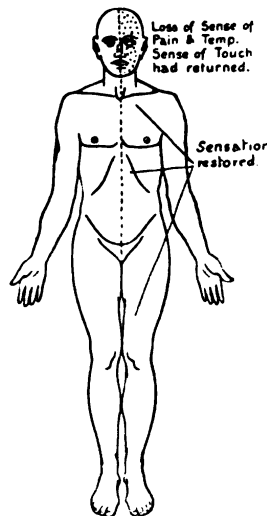


FIG. 2.

The case appears to have been one of thrombosis causing hemianæsthesia, which was already clearing up when I first saw him a fortnight after the onset. The points of interest are the depth and persistence of the anæsthesia over the face as compared with the rest of the body, and also its distribution strictly over the area of the fifth nerve. This distribution, together with the nystagmus, deafness, and loss of taste, suggests a pontine lesion as opposed to one higher in the capsular region, and as further evidence against the latter there was no hemianopia. Assuming this to be the case one must suppose, to account for the anæsthesia of the fifth nerve combined with hemianæsthesia on

the same side, that the brunt of the trouble has been borne by the fibres of the fifth after they have crossed the middle line, but just before they have actually mingled with the general sensory fibres in the fillet, which latter apparently suffered to a much less extent considering the way the anæsthesia cleared up over the trunk and limbs as compared with that over the face. Other interesting points in the case are the implication of the special senses of hearing and taste in connexion with the anæsthesia.

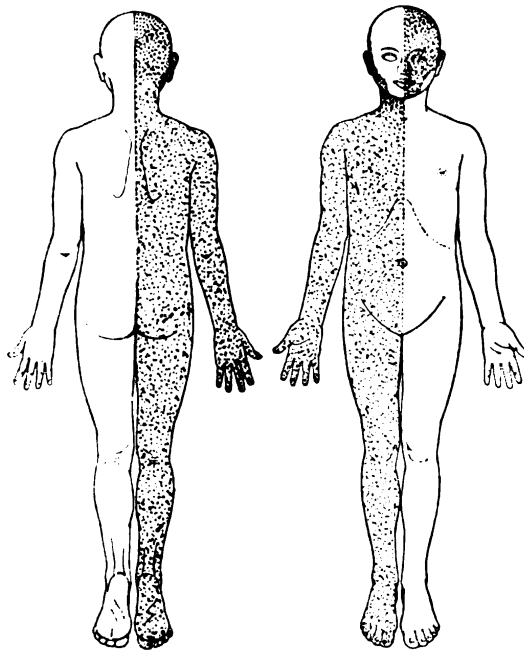
A Case of Thrombosis of the Left Posterior Inferior Cerebellar Artery followed by severe Trigeminal Neuralgia in the Analgesic Facial Area.

By WILFRED HARRIS, M.D.

J. S. M., A GENTLEMAN aged 67, in July, 1903, had a sudden stroke, with intense giddiness and inability to stand, and at the same time a prickly sensation all over the left side of his head as though hot flannels had been suddenly applied to his face and head, and he says it felt as if hundreds of needles were sticking into the scalp. There was no loss of consciousness and no obvious paralysis of any limbs, but he was put to bed, where he remained for four months. There was no vomiting or hiccough, but two days later he became unable to swallow, and he had to be fed artificially through a tube for a week. The stroke was followed immediately by slight aching around the left eye and temple, which has gradually increased, developing flashes of pain about a year later, which have gradually increased in severity, darting through the temple and left eye, and in the cheek immediately under the eye. Formerly the pain used to dart into the side of the nose, but that is now less marked, the chief sites of the spasmodic pain being over the left malar bone and above the temple. He has always been very shaky on his legs, ever since he first began to go about—four months after the stroke. Looking upwards especially causes vertigo.

I first saw him on July 27, 1909, when he was sent to me as a case of trigeminal neuralgia for treatment by alcohol injection. On examination there was no sign of hemiplegia; the knee-jerks and other reflexes were normal. Pupils and eye movements normal, except that he at once

complained of vertigo on looking upwards, especially if he were standing at the time. Sensation: Complete analgesia over the whole of the left side of the face and forehead as far back as the line joining the two auditory meati over the bregma. The prick of a pin was perceived here only as a touch, and the analgesic area included the front of the pinna and tragus, and the side of the cheek from $1\frac{1}{2}$ in. above the lower border of the jaw up to the middle line of the forehead, nose, and chin. Besides the loss to pain sensation, this area was also anæsthetic to moderate ranges of temperature, and tactile sensation was also not perfect, touches



Thrombosis of left posterior inferior cerebellar artery.

not appearing natural, though touches with cotton-wool were at once perceived. Sensation was perfect on the right side of the face and forehead, but on the back of the right side of the head behind the bregma there is similar analgesia present, including the pinna and tragus of the external ear, but not the inside of the concha. The analgesia extended on to the side of the head for a distance of $1\frac{1}{2}$ in. in front of the pinna, and also on the cheek just in front of the tragus and along the lower border of the jaw. Below, the analgesia involved the whole of the right side of the body from the border of the lower jaw downwards, the loss of

sensation being precisely similar in character to that already described on the left side of the face. He complained of intermittent flashes of severe pain mostly over the left malar bone, at the outer canthus of the left eye, and above the left temple. He always feels nervous of anyone inadvertently touching this side of his face or head, as he says a light touch may provoke a spasm of pain, though he finds that firm pressure with his hand relieves the pain when present.

The diagnosis in this case was thus comparatively easy. Besides the characteristic history of a sudden stroke, associated with intense giddiness and inability to stand for many weeks, loss of the power of swallowing for several days, prickly feeling over the face on one side, there was present the characteristic crossed dissociated sensation affecting the area of the left fifth nerve and the opposite half of the body below the level of the fifth-nerve supply, the dissociated sensation involving complete loss of painful and thermic impressions over the areas described, with no interference with tactile impressions and no subjective sensations of numbness. Hiccough was either not present at the commencement or else he has forgotten it, the stroke having occurred six years ago. Nystagmus also is not present now. In spite of the absence of these two symptoms, the clinical picture is at once recognizable as typical of thrombosis of the left posterior inferior cerebellar artery. An excellent description of this pathological condition, and of the clinical symptoms produced by it, was published by Dr. Kinnier Wilson,¹ and other good accounts were previously published by Spiller and others in America. I will show on the screen Dr. Wilson's diagram, which illustrates the area of sclerosis of the lateral region of the medulla resulting from thrombosis of the artery in question. It will be seen to contain the descending or spinal root of the fifth nerve, some of the fibres of the ninth, tenth, and eleventh cranial nerves, the decussating vestibular fibres of the eighth nerve, and the lateral fillet which probably carries ascending impressions of pain and temperature from the opposite side of the body below. The descending root of the fifth nerve appears to convey the impressions of pain and temperature of the trigeminal area of the same side, hence a lesion of the lateral region of the medulla, damaging the lateral fillet and the descending root of the fifth nerve, will cause a crossed analgesia of the face of the same side and of the body and limbs on the opposite side. In some cases the damage to the lateral fillet in the reticular formation by a thrombotic lesion is less intense than in my patient as described above, and the analgesia of the opposite side of the body may be partial

¹ *Proceedings*, 1909, ii (Neurol. Sect.), p. 52.

and incomplete. Such, for example, was the case in another patient I have seen quite recently, who, after several transient attacks of giddiness and unsteadiness during a period of three weeks, was seized suddenly with a more severe attack, in which the previous incomplete closure of the vessel evidently became complete. As a result, giddiness and unsteadiness on the left leg became very marked, and well-marked analgesia of the left trigeminal area and of the right lower extremity up to the groin appeared. For months this analgesia of the leg remained, which the patient found useful to him in playing hockey, as he was able to use that leg to stop the ball without feeling any pain from the blows. Since the lesion does not extend ventrally to the inferior olive, the pyramidal fibres escape, and, though there is generally unsteadiness on the leg of the same side, there is no real weakness or spastic paralysis of the limb.

The point of special interest in my patient, J. S. M., is the remarkable sensory development that has taken place in the analgesic facial area. Starting at the moment of the stroke with the sensation as of hundreds of needles sticking into his scalp; an aching pain followed over the whole of the left trigeminal area, which after a lapse of about a year gradually developed neuralgic characters, intermittent flashes of pain appearing over the left temple, around the outer canthus of the left eye, and inwards across the cheek on to the side of the nose, pains comparable in character and in cause to the lightning pains in tabes. These have gradually increased during the past five years, so that he now has frequent daily attacks of darting pain through the face in the regions named, though the pain does not now run into the side of the nose as it did formerly. However, as the area of neuralgic pain appeared almost limited to the distribution supplied by the second or superior maxillary division of the fifth nerve, I thought that possibly injection of the trunk of the nerve at the foramen rotundum might relieve the pain, although it was evident that there was a central lesion damaging the descending root of the fifth nerve within the medulla. The sequel, I think, shows that it is inadvisable to attempt to relieve pain due to an intramedullary lesion by means of alcohol injection or by means of resection of the nerve roots peripheral to the site of the lesion causing the pain. I performed the alcohol injection in the ordinary way, using no anæsthetic, even to the skin, as that was analgesic already. No difficulty was experienced in reaching the superior maxillary nerve-trunk, the patient being able to tell by his sensations when the point of the needle reached the nerve. About fifteen minims of 80-per-cent. alcohol

were injected slowly, with the result of causing partial tactile loss on the cheek and nose in addition to the already existing analgesia. No amelioration of the pain was, however, produced; on the contrary, the spasmodic attacks of pain since the injection have become much more severe, and no less frequent than before. I have also seen a similar result in a case of frontal herpes in a man of 50, due no doubt to a root neuritis of the ophthalmic or first division of the fifth nerve, at its origin from the Gasserian ganglion. In this case the patient complained of persistent paræsthesiæ, scarcely so much painful as annoying, over the eyebrow and forehead, where there was slight analgesia remaining from the herpes. In order to attempt relief of these abnormal sensations of which he complained, I injected the supraorbital nerve at the notch, and, though this produced deeper anæsthesia than before, the paræsthesiæ became more pronounced.

DISCUSSION

Dr. HINDS HOWELL brought forward a case the pathological examination of which he had made, and as such pathological examination was not very common in these cases of dissociated and crossed sensory disturbances, he thought it might very well be added to those which had formed the subject of the two previous papers. The patient was under Dr. Herringham's care in St. Bartholomew's Hospital, and he was indebted to Dr. Herringham for the opportunity of examining the brain. The first symptom was neuralgia, followed six months afterwards by left facial paralysis, and later by symptoms indicating a left cerebellar lesion. When in the hospital the patient was found to have a very peculiar sensory disturbance. There was complete loss of the sensations of pain, heat, and cold over the right face and the right half of the body, with the exception of the third and fourth sacral segments, which had partially escaped. On the left side there was loss of tactile sensation in the area of the fifth nerve. The corneal reflex was lost on the left side, and there was loss of taste on the left side of the tongue. After fifteen months the patient developed optic neuritis, and shortly afterwards he died. Dr. Howell showed epidiascope illustrations of the condition found on detailed examination of the pons: a tumour was seen to be lying on the left lateral aspect of the pons, in the portion of the middle cerebellar peduncle. It was partly solid and partly cystic; the solid part extended from the lower limit of the pons to a level just below the exit of the left fifth nerve. It had compressed the structure lying in the lateral part of the pons, but had not infiltrated. The cystic portion lay partly within the fourth ventricle and partly external; through the anterior portion the left

fifth nerve emerged. The sensory disturbances were probably caused by pressure on the left fifth nerve, and interference with the spino-thalamic tract at the side of the pons, in which probably ran fibres carrying pain, heat and cold impulses from the opposite half of the body and face. Examination of the pons, &c., by the Marchi method had revealed only a diffuse degeneration.

Dr. R. F. KENNEDY said that Dr. Harris's statement about trigeminal neuralgia going on as a result of a central lesion reminded him of a case he saw in 1907. It was the case of a man of about 30 years of age who was admitted to the hospital with very severe trigeminal neuralgia on the right side. He had no other symptoms or signs whatever. He was admitted in May, and remained for about six weeks, during which time nothing was done, and he then began to develop double optic neuritis of very low intensity, and, later, fifth anæsthesia of the right side. It was a very gradual onset, and about August of the same year dissociated sensory changes on the left side of the body also appeared. Intra-medullary tumour was diagnosed, but no operation was done, and the man was still living, but his neuralgia was as severe as ever, and, clinically, was of quite a trigeminal type.

Sir VICTOR HORSLEY asked whether, in Dr. Thomson's case, there was anæsthesia when the eyeball was pushed. What was the consciousness of the patient with regard to movements of the eye on the left side?

Dr. GORDON HOLMES said that he, too, had a case of thrombosis of the posterior inferior cerebellar artery similar to that which Dr. Harris had described, in which, despite all treatment, this radiating pain in the face continued, although it was not so intense as in Dr. Harris's and Dr. Kennedy's cases. He wished also to refer to the sections of the case Dr. Howell had brought forward. In attempting to determine the exact position of the conducting tracts from pathological specimens, the exact nature of the lesion was very important; there was no doubt in his mind that the actual condition of affairs in that case was due to a congenital cyst of the floor of the fourth ventricle, arising as a diverticulum from the neural tube in its development. Tumours often arose in connexion with such congenital cysts. They had then to consider the effect of a tumour on tissues which had been abnormal from birth, and in which the normal topographical relations had probably never existed; the fact that no secondary degeneration was found, and the consequent conclusion that there was no complete interruption of fibres, favour this view.

Mr. SYDNEY SCOTT said that he was particularly interested in Dr. Harris's, Dr. Thomson's, and Dr. Howell's cases on account of the proximity of the lesions to the eighth cranial nerve. He would like to know whether their notes contained details of any special examination of the "auditory system proper" or of the reactions of the "vestibular system." Mr. Scott afterwards explained that information on these points was very much needed to elucidate many of the obscure problems in connexion with the eighth nerve.

Dr. PURVES STEWART said that Dr. Harris had incidentally called attention to an important group of cases. In cases where the neuralgia was due to a lesion situated centrally to the Gasserian ganglion, operation brought no relief to the patient. He had in mind one patient, an old lady, who suffered from pain as badly as ever, in spite of the operation of Gasserectomy performed some years ago, before he saw her. He might be asked whether the ganglion was really removed; all he could say was that the surgeon provided him with a photograph of the entire ganglion after removal. Yet in that case the patient retained her pain, and it was as severe as before the operation. Unfortunately, they had no means at present of distinguishing these central cases before operation. In the case he had mentioned the patient's pain was confined to the first division of the nerve both before and after operation. Probably it was due to some central lesion in the region of the fifth nucleus in the pons.

Dr. HENRY HEAD, F.R.S., speaking with regard to Dr. Stewart's case, said that it was important to know whether in such an instance sensation of the face was carefully tested. His experience was that in cases of central disease sensation was, as a rule, altered, whereas notoriously in the ordinary true trigeminal neuralgia there was nothing that could be discovered by testing, and nothing that could really be known apart from the patient's description. In those that were central to the ganglion there was almost always a change in sensation. He admitted that the change was very various, but it had always been in the nature of "anæsthesia dolorosa"—an area of tenderness and pain, with a central portion of definitely lowered sensation. It was quite true that from the patient's standpoint there was no particular difference so far as the pain was concerned, but it was important for the neurologist not to confuse the two cases. If, on going over the face carefully with the tests, any definite lowering of sensation was discovered, the lesion was likely to be central to the ganglion, but if there was no such lowering the chances were that it was ordinary trigeminal neuralgia. He had the good fortune to see Dr. Kennedy's case because of this very striking lowering of sensation. He wished to impress a cautious policy upon every neurologist when he met with a case of supposed trigeminal neuralgia in which there was lowering of sensation of the face.

Dr. CAMPBELL THOMSON, in reply, said that in the case he brought forward no particular tests were made with regard to the auditory symptoms. The man had been deaf to some extent in both ears previously, and, though he thought he was deafer since his illness, Dr. Thomson did not in these circumstances consider the deafness a very important symptom. With regard to the other sensations, when he (Dr. Thomson) first saw the patient, he could not feel the touch of a razor at all, and his face was considerably scratched by efforts he had made to shave himself. When he was last seen, however, that particular sensibility had been regained. With regard to the eyes, he had nystagmus, but the test suggested by Sir Victor Horsley had not been carried out.

Dr. WILFRED HARRIS, in reply, said that he quite agreed with Dr. Head in his remarks about the central lesion. He made his alcohol injection with the

simple object of relieving the patient's pain, but in this respect he was unsuccessful. In the case mentioned by Dr. Purves Stewart, it appeared the latter had not had a chance of making his diagnosis because the ganglion had been taken away beforehand. Dr. Thomson had mentioned that in his case there was loss of taste on the same side as the sensory tactile losses in the area of the fifth nerve. Did not that indicate that the fillet carrying up the taste fibres had been damaged together with the other fibres of the fillet? With the lesion of the sensory tract low down in the pons taste and hearing would be involved, whereas smell and vision, which of course only came into the sensory tract higher up the back of the capsule, would not be involved.

Dr. HINDS HOWELL, answering Mr. Scott's question with regard to the eighth nerve, said that in his case there was nerve-deafness, but no special tests were applied. Dr. Gordon Holmes had spoken of the probable pathological conditions governing the case as being congenital in origin, and he was glad to have his opinion. He asked Dr. Holmes at what age the congenital cases usually made their appearance. [Dr. HOLMES: Some of the cases were those of young people, and others were middle aged.] At the same time the pathological nature of the tumour was not under discussion. His object in bringing forward the case was to show the pathological lesions associated with the crossed anæsthesia which had existed, and it was clear that one must attribute to these pathological lesions the symptoms that had been described.

Neurological Section.

April 28, 1910.

Dr. J. A. ORMEROD, Vice-President of the Section, in the Chair.

A Case of Cerebellar Sclerosis.

By GORDON HOLMES, M.D.

G. Y. is the only child of the parents; there is no similar disease in the ascendant or collateral lines. At six weeks of age she had whooping-cough, but has had no other illnesses, and never had convulsions. The symptoms were noticed only when she commenced to walk when aged 2. She has always staggered and frequently falls; it has also been noticed that the movements of her hands are irregular, and she often allows objects she carries to drop. Intelligence is said to be good.

Present state: The functions of the cranial nerves are unaffected, and there is no nystagmus. There is considerable ataxia of both the upper and lower extremities, and in walking she staggers from side to side, and keeps her feet widely separated. It is difficult to elicit the deep reflexes, but the plantar responses are of the flexor type.

The diagnosis is a cerebellar lesion which was either congenital or set in during the attack of pertussis which occurred when six weeks of age; the latter seems the more probable.

Case of (?) Olivo-ponto-cerebellar Atrophy.

By GORDON HOLMES, M.D.

M. B., AGED 64, has always enjoyed good health. He has been temperate in the use of alcohol, and there is no history or suspicion of specific infection. He has been employed as a skilled labourer in

Woolwich Arsenal. He has been aware for some years that his right hand was not quite steady, but this did not inconvenience him till about twelve months ago, when it became more tremulous and shaky; as it became worse, he had to cease work in September, 1909. For about two years he has suffered with attacks of giddiness, occasionally with a sensation of rotation of self to the right. From September, 1909, he has staggered in walking like a drunken man; this he ascribes to a feeling of being pulled to one side or the other, but chiefly to the right. His gait has gradually become worse. His left arm has been tremulous for the past two or three months. He has never had headache or diplopia, and his general health remains good.

Present state: There is no strabismus or nystagmus, and the pupils react to light and on accommodation. The optic disks are normal. The functions of the other cranial nerves are unaffected, but his facial expression seems rather fixed. There is frequently coarse tremor of large amplitude and fairly regular rhythm of the right arm when it is not fully supported, which affects the proximal and distal segments of the limb equally, and on movement there is very coarse intention tremor. The left arm, and to a less extent the right leg, show the same symptoms. The strength of the arms, especially of the right, is poor. He walks on a broad base, and reels and staggers from side to side, but more to the right than to the left. The unsteadiness is not much increased on closing his eyes. There is no sensory disturbance. The deep reflexes are brisk, but ankle-clonus cannot be elicited. The plantar responses are of the extensor type. He has not had sphincter troubles. The condition has been slowly progressive without any acute exacerbations.

DISCUSSION.

Dr. FARQUHAR BUZZARD asked Dr. Holmes what was the pathological lesion in the first case; and what grounds he had for saying it was a sclerotic process. With regard to prognosis, and the doubts which had been raised as to whether it was congenital or acquired, it was his experience that cases which displayed symptoms of this kind coming on after an infective illness tended to improve. On the other hand, he had seen two or three cases similar to the present one in which there had not been that history of following an infective fever, and in which the disease appeared to have been congenital, and those cases had always progressed towards a worse condition as the child grew older. In those cases, too, there had always been a certain amount of mental deterioration, also progressive, and in one case it was associated with progressive blindness due to retinitis pigmentosa. This experience led him to think that the acquired cases had a better prognosis than the congenital ones.

Dr. F. E. BATTEN said the first case seemed to fall in with the group of cerebellar ataxias of children, and he thought it was very difficult to say whether it was a congenital or an acquired condition. If it were congenital, he thought recovery might ensue; if acquired, improvement would almost certainly take place; but if there were progressive degeneration, the child would become more and more ataxic. He believed the present child would get almost completely well.

Dr. S. A. K. WILSON, in regard to the second case, said it was difficult to indicate the lesion definitely, because the patient had some arterial disease; and, though apparently he had not got "silver-wire arteries" in the retina, his pulse was somewhat tortuous, suggesting arterial sclerosis, which probably occurred also in the cerebellar arteries. The symptoms were partly cerebellar; the gait was suggestive in this respect; the tremor suggested mid-brain disease; there was an extensor response on both sides, which was indicative of a pyramidal lesion. The pathology was also obscure. The last reported case was in the *Nouvelle Iconographie de la Salpêtrière*,¹ but it did not give a complete history from the clinical standpoint. The pathology of it was indicated by the term "olivo-ponto-rubro-cerebellar atrophy." As Dr. Holmes said, a number of such cases had been reported, but the pathology was variable, and there were several pathological types which it was not easy to distinguish clinically.

Dr. HOLMES, in reply, said that for the first case he purposely selected the term "cerebellar sclerosis," because it did not commit him to any of the questions which Dr. Buzzard raised. If there could be an opportunity of seeing the child's cerebellum, he thought it would be found in a sclerotic condition, though whether this was congenital or acquired was doubtful. The diagnosis depended largely on the history, and in this case the history was not very good, and the child had not been under observation long enough to enable one to judge whether she was improving or not. There was no evidence to favour the view that it was a progressive disease, as Dr. Batten suggested it might be. But in support of what Dr. Batten said he remarked that he did not think Dr. Buzzard's statement, that the congenital cases remained more or less stationary and the acquired disease improved, was correct. [Dr. BUZZARD: I said the congenital ones are progressive.] Many congenital cases had been recorded which showed symptoms in early childhood and recovered completely; and, on the other hand, in certain of the acquired cases, as in Hammerberg's well-known case, little recovery took place, while in Clapton's case, one of the best recorded examples, the child was affected in its fourth year, and when aged 15 was practically quite well. Death occurred when aged 33, and the cerebellum was found much diseased. He considered, as Dr. Batten did, that probably there was disease of the cerebellum, acquired during an infective illness in the first three months of life. He thanked Dr. Wilson for the comfort he had given him with regard to his second case, and he felt one could not get much further in the matter. He agreed with

¹ Lejonne (P.) et Lhermitte (J.), *Nouvelle Iconogr. de la Salpêtrière*, Par., 1909, xxii, p. 605.

Dr. Wilson that the recorded cases of olivo-ponto-cerebellar atrophy differed somewhat from each other. On the other hand, there was in his opinion but little arterial disease in this case, and the state of the radial arteries seemed to him relatively good considering the age of the patient.

Distal Type of Myopathy.

By F. E. BATTEN, M.D.

F. S., AGED 18, the second of a family of eight—four boys and four girls—all of whom are alive except the eldest, who died of measles when aged 9 months. This case was shown at the Clinical Society in January, 1899, as “a case of progressive muscular atrophy of the peroneal type, occurring after measles.”¹

The boy was quite well till aged 3, when he contracted measles, and a few weeks later the mother noticed dragging of the left leg; seven months later the right leg became affected, and when the boy was aged 6 he began to lose power in the hands. Since then the wasting of the arms and legs has slowly progressed, but his general health has remained good, and he is still able to walk about. There is at present the most marked wasting of all the muscles below the knee and a considerable amount of wasting of the thighs. The foot is in an inverted position and the boy walks on the outer side. There is marked wasting of all the muscles of the arm below the elbow, and but little power of movement in the hands and forearms; the upper arms and shoulders are well developed. The muscles of the face and trunk are normal and well developed. The deep reflexes of the leg are absent and the plantars cannot be obtained. The abdominal reflex is active. Sensation is perfect to all forms of stimulation all over the body. There is no marked vasomotor disturbance in the legs.

Electrical reactions: No response to faradism is obtained in any of the muscles below the knee or elbow. No response to galvanism in these muscles. No polar change detected.

Dr. BATTEN said he showed the case at the Clinical Society, years ago, as progressive muscular atrophy of the peroneal type, following on measles. At that time he did not think the distal type was recognized, and it was a question whether at present there were sufficient pathological data to fully accept that idea, though he inclined to think there was. If asked for points of distinction, he would say that in the peroneal type there was considerable vasomotor

¹ *Trans. Clin. Soc. Lond.*, 1899, xxxii, p. 239.

change, that there were sensory changes, and changes in the spinal cord. In the distal type the condition started in the periphery of the limbs; there was no sensory change, and no pathological change was found in the spinal cord. In the cases described by Spiller and Dejerine, though the muscular atrophy had existed for many years, no change was found in the spinal cord. The peroneal type was practically always accompanied by definite myelopathic changes. But it was difficult to distinguish the types clinically. The present boy had wasting of all his distal muscles, while the proximal muscles were relatively well preserved. He thought the distal type was one which had come to stay, and that a number of cases of it would be forthcoming in the future.

Distal Type of Myopathy in several Members of a Family.

By F. E. BATTEN, M.D.

THE members of this family, five in number, were shown at the Clinical Society on February 28, 1902, under the title, "a familial type of paralysis allied to the myopathies and to Friedreich's disease."¹

G. K., aged 15, in 1902—the eldest member of the family—gave the following history: She had always walked slowly and been weak on her legs, but it is only during the past nine months that she has been obviously weaker on her legs. She can use her hands well. The weakness in the legs is most marked in the peroneal and anterior tibial muscles and in the extensors of the toes. There is foot-drop and pes cavus. *There is practically no wasting.* The back is straight; there is no weakness of the upper extremities or face. There is slight nystagmus on lateral deviation. The knee-jerks are absent, and the plantars cannot be obtained. No reaction to a strong faradaic current can be obtained in the muscles of either leg, not only in the muscles obviously affected, but also in those not obviously weak.

In 1907 G. K. married, becoming Mrs. B., and a female child was born, whom Mrs. B. brought to me, when aged 4 months, for examination. Nothing abnormal could be detected with the babe, which was perfectly healthy at the time; the knee-jerks were easily elicited, and there was nothing abnormal in the feet. In 1910 Mrs. B. again brought her child to see me because she walked on tip-toe. She was now aged between 2 and 3, and ran about well, but tended to walk on tip-toe. On examination, the foot was with difficulty flexed beyond the right angle

¹ *Trans. Clin. Soc. Lond.*, 1902, xxxv, p. 205.

to the leg, but there was no definite weakness. The knee-jerks were difficult to obtain, but could be obtained. The ankle-jerks were present.

Mrs. B. herself was in good general health, but her disease had progressed. Her weakness below the knee is considerable, but added to this weakness there was now marked wasting of the anterior tibial and the gastrocnemius muscles, and there was also wasting of the small hand muscles and of the forearm.

J. K., aged 19, shows a similar wasting of the muscles of the leg below the knee.

DISCUSSION.

Dr. FARQUHAR BUZZARD, discussing Dr. Batten's first case, described some years ago as peroneal atrophy, did not know why he should alter that diagnosis now. Any change was explained by the fact that the case had progressed. Considering the small amount of muscle which the boy had in his legs, he got about quite well, a point which was characteristic of peroneal atrophies. He disagreed with Dr. Batten's remark about contractures being typical of peroneal atrophy rather than myopathy; he considered the reverse to be the case. With regard to the second lot of cases, he took the view eight years ago that they were peroneal atrophy, and that view he still held. It had been several times noted in the literature of the subject that the absence of reaction to electrical currents, even in muscles which were not wasted, was characteristic of peroneal atrophy. He did not see how both these classes of case could be put into the distal category; they were all covered by the peroneal type of atrophy. If one classed as myopathies cases in which there was a marked loss of electrical response without wasting, it would be a new departure.

Dr. WARRINGTON said he would emphasize the point about the absence of electrical irritability in the non-wasted muscles in the peroneal form. It had often been pointed out, and was a marked feature in three cases he had studied.

The CHAIRMAN (Dr. Ormerod) said he understood Dr. Batten's position to be that what was called, clinically, the peroneal atrophy consisted of two types, which differed pathologically: one was a myelopathy or neuritis, and the other a myopathy. The history of progressive muscular atrophy after measles closely coincided with that of some cases, which he (Dr. Ormerod) published some time ago, and of some others published later by Dr. Donkin. In his own cases, although there was no sensory disturbance, there was an indication of the reaction of degeneration, which probably would point to a lesion in the cord or nerves.

Dr. GORDON HOLMES said his impression was that sensory changes were a prominent symptom of hypertrophic neuritis, and he believed that radiating

pains frequently occurred. Another point was as to the age-incidence of peroneal atrophy; he thought the age of onset was generally higher than in Dr. Batten's cases.

Dr. BATTEN, in reply, said he had shown the case in order to ascertain whether it was possible to distinguish the distal type of myopathy from the peroneal type; there seemed to be a consensus of opinion that it was very difficult. Dr. Buzzard had laid great stress on contractures not occurring in the peroneal type, and it was a point which Spiller made in distinguishing between the distal and peroneal types. But it was very difficult to attach so much importance to one single symptom. With regard to the electrical reactions, he understood Dr. Buzzard to say that absence of them was one of the characteristic features of the peroneal types. But he (Dr. Batten) would have thought it was one of the characteristic features of myopathy. In the simple atrophic type of myopathy or myatonia congenita the electrical reactions, considering the thinness of the skin of the child, were usually difficult to obtain, and that he looked upon as usual in the electrical reaction of myopathic muscle, and he did not think it could be used as a distinguishing point between the peroneal type and the myopathy. With regard to chronic hypertrophic neuritis, he would put cases of that condition into yet another group, putting those in one, the peroneal in another, and the distal type of myopathy in a third.

Amyotonia (Myatonia) Congenita.

By A. E. NAISH, M.B.

G. E., AGED 4 years and 11 months. For the first three months of life he was as flabby as "a lump of raw liver," and he remained very wasted and inert for the first year of life, in spite of breast-feeding and no obvious digestive trouble. The legs and the left arm were noticed to be useless from a few days after birth; the right arm was noticed to be affected from the time he began to try to use it. The feet could be dorsally flexed to touch the tibiae. He first began to sit up when aged about 3. He began to stand with assistance when aged about $3\frac{1}{2}$, and could stand entirely alone when aged about 4, and walked first alone about a month later. Family history: No other member has had any weakness of any kind.

Present condition: Shy, but intelligent. Hypotonia of all external trunk and limb muscles, more marked in the muscles of the upper extremities; the arms can be wrapped round the neck almost like a scarf, and on lifting him the shoulders nearly touch the ears. The

hypotonia affects all the muscles down to the hands and feet; the latter are flat and long, and can be dorsally flexed to within $1\frac{1}{4}$ in. of the tibia. He can perform all movements, but weakly.

On sitting, the head and upper part of the trunk are bent forward to a moderate degree. On standing, he is very uncertain at first of his balance; on getting steady he stands with a wide base, flat feet, and the natural spinal curves exaggerated rather like a medium grade pseudo-hypertrophic. He gets up off the ground like a pseudo-hypertrophic. Knee-jerks, ? just present. He is very tolerant to faradaism, and electrical reactions are much diminished.

Two Cases of a Nervous Disease of Undetermined Nature occurring in a Brother and Sister.

By GUY WOOD, M.B., and S. A. K. WILSON, M.B.

It is difficult to give a short abstract of these cases, as the symptomatology of both is somewhat complicated. The patients' father and mother were first cousins, and their mother's mother and her husband were first cousins. They have three healthy brothers and sisters, and their previous history is negative.

CASE I.

W. O., aged 31, female. Twelve years ago she began to complain of pain in the ball of her left great toe, and noticed a lump developing there, hard and tender. Seven and a half years ago a heavy lead-lined box fell on the left foot, causing further pain and swelling. She was taken to Charing Cross Hospital and operated on. The wound became infected, and took about a year to heal. The condition was diagnosed as double hallux valgus. Five and a half years ago patient noticed that she became readily tired when walking, and that she trembled with the slightest muscular exertion. She noticed weakness of her trunk muscles, and she could not rise easily. Her condition has steadily progressed. She began to drag the limbs in walking, especially the left, and noticed that her balance was upset with the greatest ease. The tremulousness has increased: it is practically constant, though worse with exertion. She can run better than she can walk; she can walk uphill backwards

much better than in the ordinary way. When standing she feels a constant tendency to go backwards. She has difficulty in rising from a chair. Her speech has become slower and monotonous, and she has noticed that she smiles or laughs very easily, especially when tired. She is said to have had precipitate micturition. She has become much thinner and weaker. No vertigo.

On examination: Face curiously expressionless and stiff. Patient is somewhat emotional. Holds neck and body stiffly; in turning head from side to side, eyes turn before head as in paralysis agitans. Hands often held in a position suggestive of paralysis agitans. Fine tremor easily seen and felt, both in arms and legs, but not a true intention tremor. On voluntary movements of limbs, the phenomenon of defective inhibition of antagonists is often exceedingly well marked. Apparently no atrophy of any particular group of muscles. Considerable weakness of flexors and adductors of hip and of extensors. When she stands with feet apart she has the greatest difficulty in bringing the legs together. Cannot turn in a seat at all easily. When limbs are being tested, muscular trembling frequently develops. No definite myotonia, apparently, but sometimes the grasps seem unnaturally strong and firm, and relaxation is not always quick. Gait, long and slow and slouching. Various sorts of "pulsion" easily obtained.

No organic signs in ordinary sense. No nystagmus. Disks normal. No true inco-ordination. No fibrillation of muscles observed. Deep reflexes lively. Abdominals brisk. Probable double flexor response; difficult to be sure.

CASE II.

F. O., aged 33, male. Twelve years ago noticed, perhaps as the result of wearing narrow boots, that the big toes were curling under the sole, and that he was beginning to "walk badly." Seven years ago, noticed weakness in his back when cycling, which used to make him lean backwards, as though to ease it. About same time became very easily tired on slight exertion. Began to "fail." His condition has steadily progressed. For the last five years the slightest muscular effort is accompanied by fine muscular tremors. He cannot walk far, not more than a hundred yards, without feeling utterly tired, but he can walk up a hill better than on the level. He can run much better than he can walk: he can run up a hill. Gait has become slow and slouching. He has the utmost difficulty in maintaining his equilibrium, like his sister. Is upset with the greatest ease. Speech has become somewhat

slow. He has recently complained of a more or less constant desire to "clear his throat," &c.

No organic signs, in ordinary sense (as in sister's case). He shows many features analogous to hers—e.g., defective inhibition of antagonists, gait, facies, muscular tremors, stiffness, instability, &c.

DISCUSSION.

Dr. HENRY HEAD, F.R.S., said he had been much interested to see Dr. Wilson's patient, as he had recently seen a case which closely resembled it. She had the same extraordinary walk and tendency to topple over, so that if care were not taken she would fall in the act of crossing an ordinary large room. Another symptom, which he did not notice in the present patient, was slight irregular movements of the head. The speech in Dr. Wilson's patient was certainly affected, and so also was the speech in his case; it was slow and hesitating, and might be termed a stupid speech. There was no nystagmus. After he had made up his mind about this case, he was told the family history. The girl's father's sister had suffered from ataxy coming on in her twentieth year, which began with inability to walk across an ordinary room and with dropping things. Three collaterals died of "locomotor ataxy," as it was termed. Two were women and one was a man. As in all of them it came on at the age of 22, it was very unlikely to have been tabes dorsalis. He regarded it as a peculiar form of family disease, and did not expect to see another case so soon. The age at which death took place varied. The father's sister died aged 54, the man at 40 or a little over, and the two women died between 40 and 50, all of the disease.

Dr. Head (answering Dr. Grainger Stewart), said the mental changes in his patient were definite, but the case was complicated by the fact that she had never been educated; though she had had a series of governesses, she had refused to learn from any of them. She had lost recognition of identity and difference, one of the earliest signs of dementia. She knew her age, but could not tell when she was born. She did not know the multiplication table, and did not recognize that 2×4 was the same as 4×2 .

Dr. S. A. K. WILSON, in reply, said he agreed that the patient was not mentally normal. He had had her under observation three years, and he thought her mental condition might be described as being "simple" or "facile." He did not think there was any dementia, and he had not noticed that restriction of memory which had been remarked on. Her mental state was not characteristic of, or analogous to, that seen in Huntington's chorea. He considered that the patient was suffering from a degenerative condition, though there were no organic signs at present. There were two sisters and a brother in the family, who were not affected. These three had fine black hair, whereas the two patients (brother and sister) were prematurely grey.

(?) Disseminated Sclerosis.

By W. J. MALONEY, M.D.

(For JAMES TAYLOR, M.D.)

E. F., FEMALE, aged 19, came to Moorfields on August 3, 1909, and consulted Mr. Holmes Spicer, by whom she was referred to Dr. James Taylor. The history was that three months before she had mistiness of her eyes, which gradually became worse. There was no diplopia. She had had giddiness for some months, and had suffered from headaches and occasional vomiting for two years. Her smell was good in both nostrils; her taste was defective on right side but good on left. Her vision only amounted to perception of light. She had some difficulty in walking; knee-jerks were very active. She had ankle-clonus and double extensor response from the sole. She was admitted to Queen Square, and went out in the autumn of 1909, but was readmitted on January 8, 1910. Her vision had very much improved: $\frac{6}{80}$ in the right eye, $\frac{6}{18}$ in the left. There was defect in the left half of the field of vision in each eye. The gait was ataxic; the articulation was rather blurred, and there was nystagmus; the optic disks had a normal appearance; the knee-jerks were very active. There was still ankle clonus and double extensor response.

The case was regarded at first as an obscure case of intracranial growth, the tumour being supposed to involve the visual tracts between the corpora quadrigemina and the occipital cortex. Further observation would seem to point to the likelihood of the case being one of disseminated sclerosis of anomalous course. The normal appearance of the optic disks is noteworthy.

Dr. MALONEY added that there was no evidence of optic neuritis.

Cerebellar Tumour.

By W. J. MALONEY, M.D.

B. S., BOY, aged 11, under the care of Dr. James Taylor; slightly built, highly excitable child, with history of headache and occasional vomiting for twelve months. January 12, 1910: Found lying on floor moaning with pain, and with back and legs rigid; for three hours he did not

seem to recognize anyone, and subsequently he lurched in walking. On examination, vision—Right $\frac{6}{6}$, left $\frac{6}{6}$; bilateral papilloedema, greater on right; eye movements normal, no nystagmus, ptosis, nor strabismus. Other cranial nerves normal; articulation slow. Slight right-side weakness of limbs and inco-ordination; in walking, right leg held stiffly abducted, right shoulder raised, right ear approximated to right shoulder; abdominal reflexes equal on two sides; plantar responses both flexor; no sensory changes. No other symptoms. Von Pirquet's cutaneous reaction negative.

Myopathy (Juvenile Type).

By P. W. SAUNDERS, M.B.

K. H., A WOMAN aged 24, in hospital under care of Dr. Tooth, complains of weakness in legs and arms of gradual onset. When aged 17, general "tiredness"; later, marked weakness in legs, difficulty in going upstairs or in rising from the ground; more than a year weakness in the arms, especially the right, and difficulty in lifting anything heavy. Four years calf muscles getting larger, thighs smaller, arms not noticed to be changed. Muscles of upper arm and shoulder show marked wasting and weakness on both sides; infraspinati a little enlarged, forearm and hand not affected. Thigh and hip muscles wasted, calf muscles enlarged, knee and hip movements very poor in power. Face and trunk not affected; no sensory changes; deep reflexes partly lost, partly absent; sphincters not affected. Gait and manner of rising characteristic of the disease. Patient has one brother, aged about 16, similarly affected.

? Syringomyelia with Brown-Séquard Syndrome.

By H. R. PRENTICE.

L. S., SERVANT, aged 29, only child of parents dead of unknown causes. Past history negative except for internal strabismus and a severe attack of influenza at the age of 12. Four years ago gradual onset of weakness and stiffness in the right foot and leg. Two years ago weakness in right upper extremity, first in extensors of right wrist, later involving the whole limb. Eighteen months ago inability to distinguish hot and cold noticed in left arm and leg. For four months drooping forwards of the head.

On admission, under the care of Dr. Gordon Holmes : Intelligent ; cranial nerves normal except for external strabismus (post-operative). Motor system : Weakness and wasting of right deltoid, biceps and extensors of the wrist ; weakness with spasticity of right triceps, pectorals and the muscles of the right trunk and the right lower extremity, causing great limitation of range of movement ; left side good range and power. Sensory system : On the left side from the level of the fifth cervical to that of the eighth dorsal segment there is total loss to all forms of sensation ; below this a relative loss. No impairment on right side. Reflexes : Biceps and supinator jerks diminished, triceps jerk exaggerated on both sides, epigastric reflex absent on left, just present on right. Abdominals present right and left ; right knee-jerk clonic, left present. Ankle-clonus on right, not on left. Double extensor response. Sphincters not affected. Marked kyphosis of cervical spine. Later there was found to be relative anæsthesia, analgesia, and therm-anæsthesia over the *right* side of the head and neck, and down the outer side of the right arm.

Syringomyelia with Total Anæsthesia.

By WILFRED HARRIS, M.D.

M. M., AGED 22, a South African, when aged 13½, while fishing off a rock, cut his left foot on a mussel shell. The wound was very painful and bled a good deal ; the foot swelled considerably, and the cut did not heal up, so that he was unable to put on a boot for two months, and the foot has remained permanently swollen and deformed since. Three months after the foot healed he developed acute loss of power in both hands, and loss of feeling to all forms of sensation on both upper extremities up to the shoulders. He thinks he may have lost the feeling on the legs at the same time, but is not sure. The sensation on the upper arms gradually recovered, leaving him unable to feel anything at all on the hands and forearms up to the elbows. The fingers of both hands gradually became contracted in the clawed position. Nothing further happened until he was aged 18, when he lost the right forefinger and top of the second finger through an injury at cricket, and also the left second toe through a poisoned wound, the fingers and toe being amputated without any anæsthetic. He says he watched the doctors doing it, feeling nothing at all. When aged 19, both legs below the knee gradually began to fail, so that in six months the right limb

was quite useless below the knee, the foot turning in. Just at that time, when aged $19\frac{1}{2}$, he suddenly lost the power in the flexors of the left fingers. Since then he has used a crutch to get about with, resting the right knee on a step fitted to it. He is perfectly certain that he could feel everything properly on his limbs before the onset of the paralysis at the age of $13\frac{1}{2}$, and that he was able to whistle, to drink, to close his eyes properly, and that in every way he was a healthy, normal boy up to that age.

Family—Has one brother and two sisters, both healthy. Knows of nothing similar in any members of his family.

Present State—Left foot: Tarsus much enlarged and arch broken down. Marked wasting of the left anterior tibial muscles and peronei. Left calf and flexors of toes fair, and left thigh and buttock normal. Right foot inverted, and hangs completely paralysed. Complete atrophy of all muscles below the right knee. The right thigh shows slight wasting of the lower portion, especially the vasti, the right thigh measuring 1 in. less than the left at the middle. All movements of the hip and knee are strongly performed. Buttocks, back and shoulder muscles normal. Arms: All the intrinsic muscles of both hands are completely atrophied, with strong flexor contracture of the two terminal phalangeal joints (claw hands). Marked wasting and weakness also of the left flexors of fingers and wrist. All other muscles in upper extremities normal. Face: Suggestive of myopathy. Cannot close eyes or purse up lips at all. Besides this paralysis of the sphincters of the eyes and lips, there is complete paralysis of the corrugator supercilii, and weakness of the frontales and zygomatici. Tongue, palate and jaw muscles normal. Eye movements and pupils normal; no nystagmus. Sensation: Total loss of all forms of sensation on both hands and forearms up to the elbows, on right foot and leg up to the knee, and on left foot and ankle. Over these areas neither touch, pain, heat, nor pressure is perceived in the slightest degree. Complete analgesia to pin-prick and loss of appreciation of heat and cold extends over the whole of the arms up to the shoulders, and over the lower extremities up to the groins and top of the buttocks, including the genitals. The whole of the upper face, forehead, and top of head are also analgesic as far back as the crown, including the ears, though just behind these the sensibility is slight; all forms of sensation on the conjunctivæ, lips, tongue, and inside of mouth are perfect. The neck is partially analgesic. All forms of sensation on the trunk below the clavicles are normal. Reflexes: Right knee-jerk feeble, left scarcely obtainable. Achilles jerks and plantars unobtainable. Sphincters normal.

DISCUSSION.

Professor PETREN (Stockholm) said he believed this was a case of leprosy. There had been several autopsies in Sweden on similar cases in which syringomyelia had been diagnosed, but they had turned out to be leprosy. If this were a case of syringomyelia, it was remarkable that the loss of sensation should have corresponded only with the cervical and lumbar enlargements, and yet be so severe. For, notoriously in syringomyelia, if the extent of the loss of sensation corresponded to the two enlargements, it was not so severe as to attack every form of sensation. That, in his view, pointed much more to a peripheral lesion. The loss of sensation in the head was in favour of leprosy rather than against it. The nerves at the back of the neck, particularly in the sterno-mastoid region, could be felt; the peroneus externus and the facial nerves could also be felt. The eye condition was also in favour of leprosy; it was characteristic, and was due to affection of the facial nerve. It was because of this facial weakness that the patient could not close his eyes. The history of the manner of development corresponded neither with that of syringomyelia nor leprosy; and it was possible that the story, by frequent repetition over a long time, had been made somewhat "diagrammatic." He understood that leprosy occurred in South Africa.

Dr. PRENTICE said that the case reminded him of one which was in the National Hospital two years ago, under the care of Dr. Aldren Turner. In that instance at first a diagnosis of syringomyelia was made, owing to the fact that while the sensory loss was of a peripheral type in the upper and lower limbs, there was dissociated anæsthesia on the trunk. Attention being drawn to the presence of some slight thickening of the ears, a piece was removed from one of them for microscopical examination. Sections showed the presence of leprosy bacilli, and the diagnosis of leprosy was abundantly confirmed at the autopsy.

Dr. FOSTER KENNEDY said that Dr. Risien Russell had lately had a patient in the National Hospital who showed a similar condition, and the state of whose hand was precisely that met with in syringomyelia. The sensory condition might easily pass for such. She had keratitis, which was thought to be due to leprosy; and he had heard that her daughter had developed similar symptoms. She had a thickening of the ears, but no tissue was removed, so the lepra bacillus was not found, but the skin affection of the daughter afterwards seemed to put it beyond doubt.

Dr. WILFRED HARRIS, in reply, said that he had at first considered leprosy as a possible diagnosis, but had discarded it for several reasons. There was no hypertrophied tissue present, and the nerves which should be hypertrophied in leprosy, such as the ulnar, could not be felt at all. Professor Petren stated that the external popliteal and facial nerves were enlarged, but neither he nor others who had examined the case with him could feel them. Certainly the patient's ulnar and external popliteal nerves were much less easily palpable than his own corresponding nerves were, and he certainly had not got leprosy.

The man was intelligent and was quite certain that the main symptoms had developed in a week, which was quite inconsistent with a diagnosis of leprosy. In reply to Professor Petren's remark that several cases diagnosed in Sweden as syringomyelia had proved later to be leprosy, he would remind him of the cases of Morvan's disease in Brittany, which were diagnosed during life as leprosy but were proved at the post-mortem to be cases of syringomyelia. Some seven or eight cases of syringomyelia, with total anæsthesia, had been published with autopsies, and in each the central gliosis was found to have destroyed the posterior columns.

Myoclonus with Spasm of the Tongue.

By WILFRED HARRIS, M.D.

F. G., AGED 53, labourer, has noticed, during the last six or seven months, constant more or less rhythmic movements of his jaws and tongue, and also of his left foot. These are present all day, and cease only during sleep. He has no pain, and he feels otherwise in good health. He cannot give any reason for their onset, and he knows of nothing similar in any members of his family.

Present condition: He is of an extremely dark complexion, with some cyanosis of the face and lips. He says his skin has always been of this colour, and he ascribes it to outdoor exposure. The heart is somewhat enlarged, and there is present a certain degree of compensated mitral regurgitation. There are constant, almost rhythmic movements of the jaws, resembling chewing movements, the cheeks appearing sunken, owing to his being quite edentulous. When the mouth is opened the movements of the jaws cease, but the tongue movements continue. The tongue is seen to be curled, and the left side raised, most of the tongue movements being directed towards the right side. Articulation does not appear to be interfered with.

Besides the tongue and jaw movements there are irregular clonic contractions of the dorsiflexors and extensors of the left ankle and toes, and of the flexors and extensors of the fingers of the right hand. When his attention is directed towards the movements of the left foot, the ankle movements cease, and the only remaining contractions occur alternately in the extensors and flexors of the toes. As soon as his attention is again distracted the ankle movements begin again strongly. There is no muscular wasting, the reflexes and sensation are all normal, and nothing further abnormal is to be made out from examination of the nervous system.

Neurological Section.

June 23, 1910.

Dr. J. A. ORMEROD, Vice-President of the Section, in the Chair.

Acute Degenerative Changes in the Nervous System, as Illustrated by Snake-venom Poisoning.

By WALTER K. HUNTER, M.D.

FOR some years past I have been engaged, in conjunction with Major Lamb, I.M.S., on an investigation into the action of venoms of different species of poisonous snakes on the nervous system, and our results have appeared from time to time in the *Lancet*,¹ in a series of six papers in all. This work has furnished me with a large number of microscopic sections illustrative of acute degenerative changes in ganglion cells as a result of toxic poisoning, and I have ventured to think some of these of sufficient interest to permit of me bringing them before the notice of this Section. In doing so I wish to express the debt I owe to Major Lamb for so freely placing at my disposal the tissues from the animals killed in the course of this inquiry; for their histological examination I myself am entirely responsible.

Monkeys were the animals used in these experiments. They were injected with varying doses of one or other of the venoms; the toxic symptoms were observed and any corresponding changes in the nervous system subsequently investigated. Perchloride of mercury was the fixing agent almost invariably adopted, and the sections were stained with thionin according to Nissl's method. Thirty-nine monkeys were in

¹ *Lancet*, 1904, i, p. 20, ii, p. 518, 1146; 1905, ii, p. 883; 1906, i, p. 1231; 1907, ii, p. 1017.

this way examined, and by way of controls similar sections were prepared from four normal monkeys.

The following were the venoms employed: (1) Cobra venom, (2) venom of *Enhydrina valakadien* (common seasnake), (3) venom of *Bungarus cæruleus* (common krait), (4) venom of *Bungarus fasciatus* (banded krait), and (5) venom of *Daboia russellii* (chain viper). The first four of these venoms had without doubt a direct action on the central nervous system, and the symptoms produced by any one of them had much the same characters as those produced by any one of the other three. Large doses of any of the four would cause generalized convulsions, with death supervening in three to four minutes. But with smaller doses death would be delayed several hours, and there would be no stage of preliminary excitement; indeed, the first symptom was now one of drowsiness. Paralysis of the extremities would next appear, more marked in the hind than in the fore limbs. Then the paralysis would become so marked that the animal could not move. Respiration is next involved, being first slow with inspiratory effort, and later it ceases altogether. Whilst, therefore, there is a widespread paralysis with all these venoms, the cause of death is paralysis of respiration.

The similarity in action of the first three venoms in particular was specially marked, for it was almost impossible by observing the symptoms to say with which of the three the animal had been injected. It has been determined, however, that there are some minor differences. For example, the venom of the common krait has a distinct action on the vasomotor centre, causing paralysis of this centre and marked fall of blood pressure, whilst cobra and sea-snake venoms have no such effect. Again, cobra venom has a considerable action on the vagal cardio-inhibitory centre, whilst the common-krait venom has only a slight action and sea-snake venom none at all. The longest time that any animal lived after receiving a minimal lethal dose of any of these three venoms was: with cobra venom, six and half hours; with sea-snake venom, six and three-quarter hours; and with common-krait venom, sixteen and a half hours.

With the fourth venom, that of the banded krait, the symptoms differed in some respects from the other three. With a large dose there were convulsions and death in a few minutes, due in this case to intra-vascular thrombosis; with a smaller dose the symptoms were those of cobra-venom poisoning—that is, paralysis of the limbs and later of respiration, only with the krait venom death was much longer delayed, one of our animals not dying till forty-four hours after injection. But

with a still smaller dose death might be delayed for twelve days. When this was so there would be an interval of two to six days when there were no symptoms except that the animal seemed depressed and off its food. Then it would begin to emaciate till the emaciation assumed the characters of muscle atrophy with corresponding muscle weakness; and before death, in some of the animals, there seemed to be more or less complete paralysis. The symptoms suggested, therefore, an acute and progressive muscular atrophy.

The fifth venom investigated—that of *Daboia russellii*—showed little action on the nervous system. In large doses it produced an extensive intravascular thrombosis, the animal dying in a few minutes. With smaller doses the animal might live for several days, and then there is no such thrombosis, and no paralysis of the limbs or of respiration. Death, indeed, seemed to be due to cardiac syncope: Rogers holds that it depends on a vasomotor paralysis of central origin.

That, then, is a brief résumé of the symptoms produced by the several venoms, and now we come to consider the histological changes met with as a result of their action on the nervous system. And first in regard to *Daboia* venom. Briefly, it had no recognizable action on the motor ganglion cells of the cortex, pons, medulla or cord, even in an animal that lived for sixty hours after injection of the venom. The suggestion that death was due to vasomotor paralysis made us pay very special attention to the “vasomotor area” in the medulla. And so we examined this area by means of serial sections in four of the monkeys. In two the sections were cut transversely, and in two in longitudinal direction. For comparison serial sections were cut through the corresponding areas of four normal monkeys. But careful comparison of the sections from the normal monkeys with those from the animals killed with the venom failed to show in the latter any certain signs of even an early degeneration in ganglion cells. Histological examination, therefore, was negative as regards giving evidence of *Daboia* venom having a selective action on the ganglion cells in that part of the bulb within which the vasomotor centre is supposed to be situated. The sections, then, from the monkeys killed with *Daboia* venom fail to supply us with specimens of degenerative lesions in ganglion cells; yet they are not without their value, for they serve as controls with which to compare the sections from the animals killed with the other venoms.

Now as to the other four venoms, we have just seen that they each produce symptoms of a widespread action on the central nervous

system; and histological examination confirms this by showing a correspondingly extensive degenerative change in the motor ganglion cells of cortex, pons, medulla and cord. The type of cell degeneration varies little with these several venoms, only they do not all produce their histological effect with equal rapidity. Thus, in an animal dying one and a third hours after injection of sea-snake venom, the ganglion cells showed undoubted chromatolytic changes; whereas in the animals dying two hours after cobra venom, or four and a half hours after the venom of the common krait, their cells presented little abnormality. Speaking generally, however, the longer the animal lived after injection, the more marked were the histological changes, and hence within certain limits these changes were in inverse proportion to the dose of the venom. In other words, the histological changes in the cells seemed to depend less on the quantity of venom than on the length of time it had to act. The action of certain venoms, too, seems much more enduring than that of others. For example, after injection of a small dose of cobra venom, if the animal does not die within two to three days at the longest, recovery invariably takes place, and recovery, too, from any paralysis that may have developed is very rapid. With the venom of *Bungarus fasciatus*, on the other hand, symptoms need not appear till six days after injection, and death may be delayed till six days later still. If, then, the changes in the ganglion cells are in proportion to the length of time the animal lives, it was thought that by studying the cells in animals dying at varying intervals, one might be able to trace the stages of this degenerative process, for we have sections showing changes in the cells from animals dying one and one-third hours, four hours, six hours, six and three-quarter hours, ten hours, twelve hours, fourteen and a-half hours, forty-four hours, three and a-half days, five days, six days, eight days, and ten days after injection. It is, however, only generally true that the changes in the cells are in proportion to the time the animal lived, for the degenerative process in the animals that lived for days was rather less acute than in those that died in a few hours; and so one finds examples of more extensive disintegration of the ganglion cell in those animals that died in six to seven hours than in the monkey that lived for ten days.

The process of degeneration, as far as can be made out, is something as follows (see Plate, figs. 5 to 26): In the first place the cell stains with basic dyes more intensely than normal, and one was forced to recognize that the sections from the paralysed monkeys took longer to decolorize than those from the control animals. This staining, while

deeper, is possibly rather more diffuse than normal, and it gives the appearance of deeply-stained tigroid bodies in a less deeply-stained protoplasm. The tigroid bodies next appear as if they were being dissolved in the cell protoplasm, and they suggest the idea of pieces of metal being acted on by an acid medium. The chromatic granules are now smaller than normal, but there is never the dust-like chromatolysis that one sees in the more chronic degenerations; neither is there enlargement nor globular deformity of the cell itself. The granules and diffuse staining next disappear, and leave a skeleton cell with its margin, reticulum, and nucleus well differentiated, though rather deeply stained. The cell reticulum has sometimes a granular appearance, but in any case it stains well with the basic dye. Later on the cell stains much more faintly, and is then the typical "ghost" cell. During almost any of these stages vacuoles may appear in the body of the cell, and its margins frequently become indented, as if little pieces had been bitten out of them. Finally, the cell begins to disintegrate, and portions entirely disappear, leaving behind little more than the nucleus, with perhaps some adherent stroma attached to it. Vacuolation, as well as disintegration of the cell, is seen mostly in the pale (ghost cell) stage, but in the most acute degenerations it is frequently met with while the cell is still deeply stained. During the whole of this degenerative process the nucleus, at least in a large proportion of the cells, seems to be little affected otherwise than is shown by a varying intensity and diffuseness of its staining. It remains central in the vast majority of the cells, and shows little change in size or shape. The nucleolus, too, shows little change. In the more rapid degenerations, for instance, with cobra or sea-snake venom, the Nissl granules seem to be more or less uniformly affected throughout the whole cell, and hence it could not be said that the chromatolysis was either "peripheral" or "perinuclear." With the more slowly-acting venoms, on the other hand—e.g., *Bungarus caeruleus* venom—a proportion of the cells suggested that the chromatolysis was, to begin with, perinuclear, and that it extended from thence outwards, for in these cells the perinuclear area was devoid of granules, whilst granules were still present at the periphery of the cell.

There was nothing specially distinctive in the histological appearances in the monkeys killed with the venom of banded krait, and which lived from five to ten days after injection. Such a length of time should permit of extreme changes developing in the ganglion cells; also it might almost give time for trophic symptoms to appear in the muscles and other tissues. General emaciation was very noticeable in these

animals, some of them losing from 20 to 25 per cent. of their weight in a few days; but it is very doubtful if this emaciation can in any way be related to the changes in the anterior horn cells. We do not know on what day the degeneration in these cells first appeared, but in Monkey No. 19 signs of paresis were not noted till the fifth day, and paralysis was not complete till two days later. Neither were we able to show that the venom was having a selective action, affecting certain ganglion cells before others. In all the animals killed with this venom practically all the cells in the anterior horns of the cord were entirely free from granules. Some cells were faintly stained, and some deeper stained, skeletons, and many showed considerable disintegration of their protoplasm. It could not, however, be determined that there was any actual loss in the number of cells in the anterior horns, and there was little difference in the appearances of the cells in the monkey that died in ten days from those of the animal that died in three and a half days after injection. It is, of course, difficult to be certain as to what is the change in the cell that constitutes loss of function. In some of the monkeys killed with cobra venom—e.g., Monkey No. 10—there was definite paralysis, but no recognizable structural change in the ganglion cells of the motor cortex or cord; and, on the other hand, it is recognized that there may be almost complete loss of Nissl granules without paralysis or other loss of function.

We attempted, but not with much success, to get some observations on this subject by examining the ganglion cells of monkeys that had recovered after being paralysed. The first of these monkeys was injected with cobra venom and it showed signs of paralysis in two hours ten minutes. Antivenomous serum was then given intravenously, and paralysis was still present fifty minutes later. The dose of antivenom was repeated and one and a half hours later all signs of paralysis had gone. The animal was at once killed. On examining the ganglion cells of the motor cortex and anterior horns of the cord it was found that quite 50 per cent. of the cells showed some deficiency of their Nissl bodies, and many of them had these bodies fragmented, giving a much finer granulation to the cell protoplasm than was normal. In some cells the granulation was quite dust like, but all gradations were found between these and the cells with Nissl bodies of normal size. Generally, the cells had the appearance of a much more chronic chromatolysis than seemed to be present in other cases of cobra poisoning; also there was little diffuse staining of the cell, and the staining was not so intense as in the cases not treated with antivenom. Two other monkeys

were similarly injected, first with venom, and when paralysis was complete with antivenom one animal was killed twenty hours and the other forty-four hours after signs of paralysis had disappeared. In both of these the ganglion cells were practically normal. The Nissl bodies in a proportion of the cells were possibly slightly deficient in number and not quite so large as in a normal animal, but, on the other hand, some of the adjacent granules seemed to have become fused together, forming masses of chromatic material considerably larger than normal. In none of the cells was there any appearance of fragmentation of granules, as in the first animal.

In these three monkeys I take it that there had been at least slight chromatic changes in the ganglion cells before the antivenom had been injected, for other animals dying paralysed in two hours after injection with the same venom showed such slight changes. The second animal would seem to show that the cells return to their normal within twenty hours after the paralysis disappears. But it is doubtful what interpretation one should give to the appearances in the first of the three. It may be that they represent a regeneration of the chromatic material. This, however, is not probable, for though changes had almost certainly commenced in the cells before the antivenom had been given, these changes must have been slight and not nearly so marked as those seen in the animal after recovery from its paralysis. It is more likely that the antivenom inhibited the venom and so modified the degenerative reaction in the cell, for the appearances were such as one associates with a much more chronic chromatolysis than that produced by ordinary cobra venom.

The *connective-tissue elements* (glia cells) of the grey matter seem to play an entirely secondary part in the degenerative changes produced by snake venoms. These glia cells may be slightly increased in number round the ganglion cell in its earliest stages of chromatolysis, but this increase is not in the least considerable, and it is not till vacuolation comes on and the cell begins to disintegrate that they are seen to cluster definitely round the disappearing ganglion cell. During this later stage, and sometimes also at an earlier stage, the glia cells are found indenting the margins and sometimes they are right inside the body of the cell. It is doubtful if there is any definite increase in the number of the glia cells throughout the rest of the grey matter, and they did not seem more numerous in animals living eight to ten days than in those dying in a few hours. The individual cells surrounding the effete ganglion cells are distinctly increased in size as compared with ordinary glia cells,

and there seems little doubt that they are derived from pre-existing glia cells and that they are neuroclastic in function.

Nerve fibres from the peripheral as well as from the central nervous system were stained in various ways so as to demonstrate any change in their myeline or axis cylinders. The peripheral nerves examined were the vagus in the cervical region, the median near the elbow, and the posterior tibial from the lower leg. In all the animals the sections were stained (1) with osmic acid, according to the method of Marchi, or that of Busch; (2) with thionin or Congo red, according to Nissl's method; and (3) with hæmatoxylin and eosin, or van Gieson's method. In none of the animals was there a genuine degenerative reaction with the Marchi or Busch method. In some sections there was slight "blackening" of some of the fibres, but I could not satisfy myself that this was not an artefact, and in any case the axis cylinders in these fibres were shown to remain intact. There was not even a definite "Marchi reaction" in the monkeys that lived for eight and ten days after injection of the *Bungarus fasciatus* venom. It is, of course, difficult to know what effect chromatolysis in a ganglion cell has on its nerve fibre, but in any case the absence of a secondary degeneration in the nerve fibres in these animals can be explained on the supposition that the degenerating ganglion cells had not lost their trophic functions till, say, forty-eight hours or so before the death of the animal. With the other stains there seemed to be, in a number of the monkeys, a slight increase of the internodal nuclei of the peripheral nerves. This was perhaps most definite in the animals that lived longest—for example, in the monkey that lived for ten days. With the view of showing the earliest recognizable degeneration in myeline and axis cylinders, nerve fibres from certain of the monkeys—those killed with sea-snake and with common-krait venom—were also stained by Donaggio's method. With this stain (see Plate, figs. 27 to 31) it seemed definitely shown that a considerable proportion of fibres, of the central as well as the peripheral nervous system, gave the appearances of an early degeneration. These fibres stained much more intensely and resisted much longer the decolorizing agents than do the nerve fibres of a normal animal. In the brain and spinal cord the deep staining seemed to affect chiefly the axis cylinders. These structures, in addition to their deep staining, were in places wavy in outline, and occasionally showed slight swellings so as to form at intervals spindle-like thickenings. But the myeline also retained the stain more tenaciously than normal, though less uniformly than the axis cylinders. This patchy staining

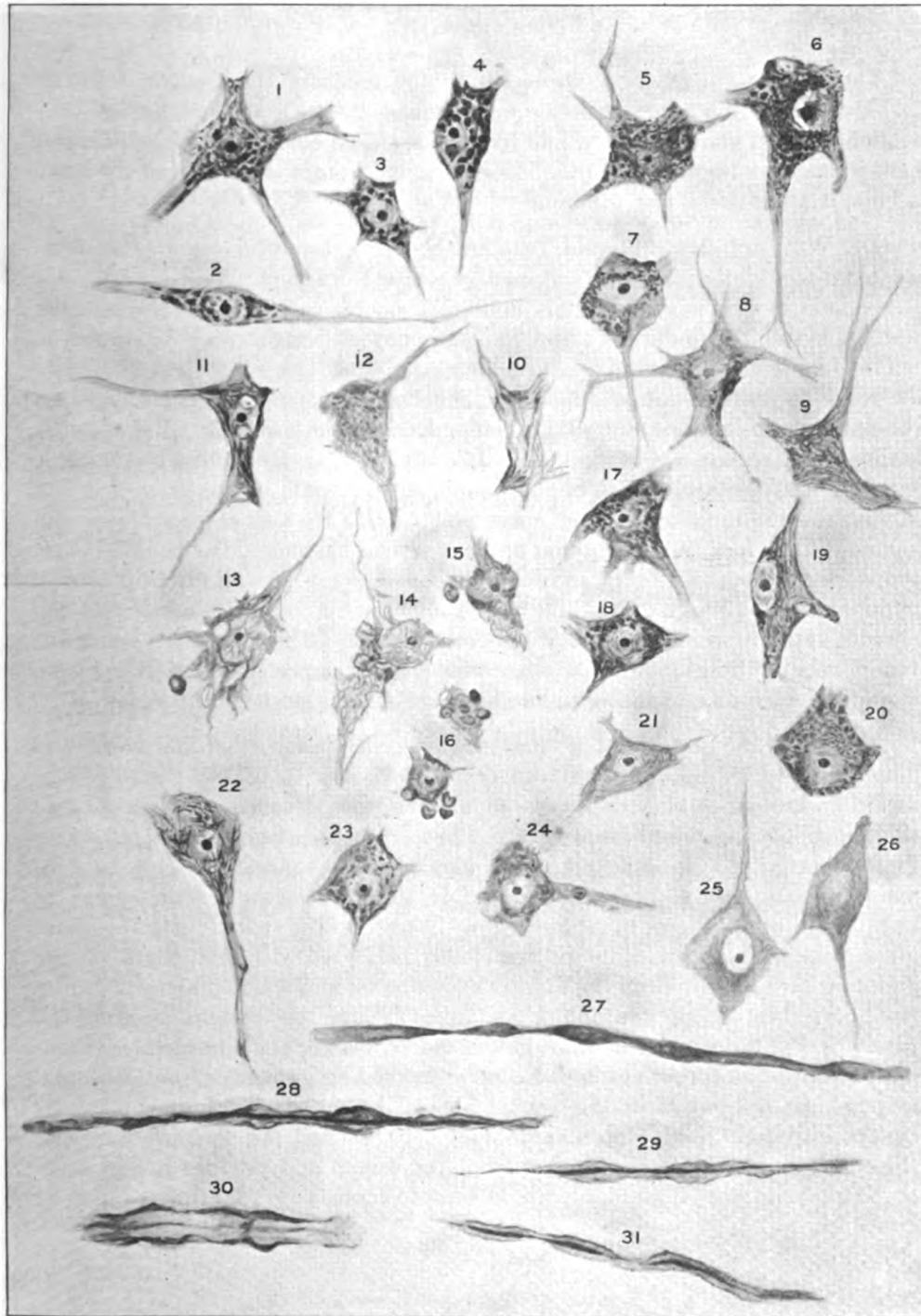
of the myeline seems to give the fibre an uneven outline, constrictions alternating with apparent swellings. In some of the fibres the myeline and axis cylinders stain the same colour and with the same intensity, so that one could not be distinguished from the other, the two seeming to have merged into one. In the peripheral nerves and nerve-roots something of the same appearances were to be seen; but as a rule the deep staining of the axis cylinders was less in evidence, and what stained most intensely seemed to be a fragmented myeline, as represented by deep-stained granules, most often arranged at the margin of the fibre, but sometimes scattered through its whole diameter. This granular appearance was most marked in the monkeys that lived for the longer periods, whilst in Monkey No. 39, which lived for one and a third hours, there was less granulation visible and more of the deep-stained axis cylinder. I take it, then, that the first change in the nerve fibres in snake-venom poisoning shows itself by a deeper staining of the axis cylinders, and that later this quality passes to the myeline, which later still may show some signs of fragmentation. There is therefore an early degeneration in these nerve fibres, but it seems to be chiefly chemical in nature, for there was little structural alteration to be demonstrated. The axis cylinders are in places somewhat swollen, and the myeline uneven in outline, with rarely some granular disintegration. But there was no definite Marchi reaction and certainly no evidence of Wallerian degeneration, even in the animals living for eight and ten days after injection.

Nerve terminals were examined in monkeys killed with Daboia venom, in those killed with sea-snake venom, as well as in those killed with common-krait venom. The nerve terminals were fixed and stained in osmic acid. With the first of these three venoms the terminals were practically normal. With the second a considerable proportion of the fibres were also normal, but others had their myeline rather wavy in outline and segmented at frequent intervals. In a few other fibres still the myeline seemed entirely fragmented and to be represented by small granules occupying the whole diameter of the fibre. With the venom of the common krait, especially in Monkey No. 31, which lived for sixteen and a half hours, the same sort of granular appearance was to be seen, but a larger proportion of fibres was affected. Some few fibres stained diffusely, showing no differentiation of myeline or axis cylinder, and they looked as if their contents were in solution. The nerve terminals would seem then to show in a proportion of their fibres the appearances of a commencing degeneration.

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DESCRIPTION OF PLATE.

- Figs. 1, 2, 3, and 4.—From monkeys killed with Daboia venom. These cells may be regarded as being normal.
- Figs. 5 and 6.—From Monkey No. 35, which died in six and three-quarter hours after injection of sea-snake venom. Shows some fragmentation of the Nissl granules and the whole cell rather diffusely stained.
- Fig. 7.—From Monkey No. 36, which died in six and three-quarter hours after injection of sea-snake venom. Cell paler with disappearance of the Nissl granules from a part of the cell.
- Figs. 8, 9, and 10.—From same monkey as fig. 7. Show deep-staining reticulum, but almost complete loss of granules. In fig. 8 there is a neurophage at the margin of the cell.
- Fig. 11.—From Monkey No. 37, which died six and three-quarter hours after injection of sea-snake venom. Shows loss of Nissl granules as well as vacuolation of nucleus.
- Fig. 12.—From same animal as fig. 11. Shows pale cell with no appearance of nucleus or limiting membrane.
- Fig. 13.—From Monkey No. 38, which died four hours after injection of sea-snake venom. Shows a skeleton cell in the process of disintegrating; there is vacuolation and a neurophage visible.
- Fig. 14.—From same monkey as fig. 13. Shows later stage of disintegration.
- Figs. 15 and 16.—From same monkey as figs. 13 and 14. Shows little more than nucleus surrounded by neurophge cells.
- Figs. 17, 18, and 19.—From Monkey No. 39, which died in one and a third hours after injection of sea-snake venom. Shows Nissl bodies breaking up and disappearing; vacuolation in fig. 19.
- Figs. 20 and 21.—From Monkey No. 33, which died in ten hours after injection of common-krait venom. Fig. 20 shows fragmented granules; in fig. 21 the granules have disappeared.
- Fig. 22.—From Monkey No. 31, which died in sixteen and a half hours after injection of common-krait venom. Shows cell deeply stained, with sort of reticulum, but no granules.
- Figs. 23 and 24.—From Monkey No. 18, which died in six days after injection of banded-krait venom. Fig. 23 shows a few granules; in fig. 24 they have almost disappeared, and there is a vacuole and a neurophge cell.
- Figs. 25 and 26.—From Monkey No. 20, which died in ten days after injection of banded-krait venom. Show "ghost" cells without granules.
- Fig. 27.—From Monkey No. 37, which died six and three-quarter hours after injection of sea-snake venom. Shows nerve-fibre irregular in outline with axis cylinder and myeline not differentiated.
- Fig. 28.—From Monkey No. 36, which died in six and three-quarter hours after injection of sea-snake venom. Shows axis cylinder deeply stained and differentiated from foam-like myeline.
- Fig. 29.—From Monkey No. 39, which died one and a third hours after injection of sea-snake venom. Shows axis cylinder deeply stained, with myeline faintly indicated.
- Figs. 30 and 31.—From Monkey No. 32, which died in twelve hours after injection of common-krait venom. Fig. 30 shows both axis cylinder and myeline; fig. 31 shows spindle-shaped swelling of axis cylinder.



Acute degenerative changes in the nervous system, as illustrated by snake-venom poisoning.

DISCUSSION.

The CHAIRMAN (Dr. J. A. Ormerod), in the name of the Section, thanked Dr. Hunter for his paper and the work it had entailed, and for the beautiful sections he had shown. He would like to hear from some pathologist how the cells in such a case differed in their degeneration from that produced by other agents, if there were any difference.

Dr. WILFRED HARRIS said that Dr. Hunter's beautiful sections demonstrated a fact which had been accepted clinically for a long time—namely, that the venom of the cobra and other colubrine snakes caused death through the nervous system. He did not claim to be a micro-pathologist with regard to the nervous system, but the sections demonstrated that the cells were distinctly altered. In cases of intoxication with the Daboia venom, the cells appeared comparatively normal. He asked whether Dr. Hunter could say what was the action of antivenene upon the nerve cell, if any. Dr. Hunter seemed to think it acted in destroying the action of the venom, but he would like to know if it had any restorative function on the nerve cell itself. He also asked whether the venom of the viper had any action on the nervous system. He believed that the work of Leonard Rogers showed that the viper venom, if given in certain minimal doses, caused death, not by thrombosis, but by some action on the nervous system resembling that of the cobra venom. The fact that sea-snake venom was ten times more toxic than even cobra was probably due to it biting in water, which made it liable to lose a considerable quantity of its venom.

Dr. HUNTER, in reply, said that the experimental part of the work was done in India, by Major Lamb, who sent the tissues to him in paraffin. In regard to the mode of action, the venom must get attached in some way to the ganglion cell, and the antivenom when injected, without doubt, liberates the venom from this attachment. Apparently it was not a very close attachment, not like that of tetanus toxin, where the anti-tetanic serum seemed to have very little effect in the way of liberating the toxin. In the cases under notice, an animal could be completely paralysed with, e.g., cobra venom, and after giving antivenene the animal would be up and walking about within a few hours. On killing the animal immediately after the disappearance of the paralysis, the ganglion cells were not found to be perfectly normal. Examination showed that these cells had a more marked and a more chronic chromotolysis than one found in the animals killed in the stage of the paralysis before antivenene had been given. The view he had put forward was that the antivenene must modify the action of the venom and produce a less acute chromotolysis than that met with in snake-venom poisoning unmodified by antivenene.

An Examination of the Blood Serum of Idiots by the Wassermann Reaction.¹

By H. R. DEAN, M.B.

THE examination of the blood serum of idiots by the Wassermann reaction has been the subject of several papers. Raviart, Breton, Petit, Gayet, and Cannac [6] examined 246 cases, of which 76 were found to give a positive reaction. Kellner, Clemenz, Bruckner, and Rautenberg [2] examined 216 cases, of which 13 gave a positive reaction by Stern's method and 9 gave a positive reaction by the original Wassermann method. To the 13 cases must be added 3 cases which were deficient in complement and were found to be positive by the original method. H. Lippmann [4], working in Wassermann's laboratory, examined 78 cases at Uchtspring, and obtained a positive reaction in 7 cases—that is to say, 9 per cent. An examination of the cases at the Dalldorf Asylum gave a result of 13·2 per cent. Lippmann also examined 77 cases by clinical methods, and decided that 40·2 per cent. showed signs of congenital syphilis.

At the suggestion of Professor Wassermann, I investigated the inmates of the Wilhelmstift, an asylum for idiots at Potsdam. The inmates were all children or young adults, and the majority were cases of simple idiocy or imbecility. The method was as follows: (1) All the cases were examined for evidence of congenital syphilis; (2) a sample of blood was taken from the arm vein and tested by the Wassermann reaction; (3) subsequently the cases which had given a positive reaction were submitted to a further clinical examination. The serum test was carried out strictly in accordance with the original method, and watery extract of congenital syphilitic liver was used as antigen in all cases. The extracts and the hæmolytic system were carefully standardized by titration, and in the actual test adequate controls from well-authenticated cases were invariably employed. No test was considered positive when more than the slightest trace of hæmolysis could be detected; 330 cases were examined, of which 51 gave a positive reaction—that is to say, 15·4 per cent. Among the 51 cases which gave a positive serum reaction, 7 were found which had definite signs of syphilis, and 3 or 4 in which syphilis might have been suspected but not with certainty.

¹ From the Serological Department of the Royal Institute for Infective Diseases in Berlin.

diagnosed. There were 2 cases with definite signs, which gave a negative reaction. That is to say, among the 330 patients were 9, or including the doubtful cases 13, which from physical signs and symptoms would have justified the diagnosis of syphilis. The results in detail were as follows:—

	Cases examined	Positive
Simple idiocy of all grades	287	44
Congenital spastic diplegia (Little's disease)	15	1
Marked hydrocephalus	14	4
Epilepsy	1	1
Microcephalic cases	4	—
Mongols	1	—
Deaf and dumb	7	1
Progressive muscular dystrophy, with mental symptoms	1	—

All the cases which gave a positive serum reaction were subsequently very carefully examined with the object (1) of discovering any sign of syphilis, which had been previously overlooked, and (2) of detecting any symptom or group of symptoms common to all the positive cases. Of the positive cases, one was subject to epileptiform convulsions and showed slight choreic movements, one had strabismus and nystagmus, one had a right-sided hemiplegia, one had spastic diplegia and conformed to the type of Little's disease, one was a deaf-mute, two were aphasic. Among the remaining cases I was unable to detect any evidence of a local lesion.

An examination was made of the cerebrospinal fluid from 12 cases, which had given a positive serum reaction. In only one case was a positive reaction obtained. I also obtained for examination specimens of serum from the parents of 10 of the positive cases. The results were as follows:—

Initial of patient's name	Age of patient	Result of examination of parents' serum
B. ...	9 ...	Father positive
		Mother ..
Har. ...	15 ...	" "
Kr. ...	11 ...	Father ..
		Mother ..
M. ...	11 ...	Father ..
V. ...	13 ...	Mother ..
O. ...	11 ...	Father negative
Sc. ...	16 ...	" positive
R. ...	12 ...	" negative
N. ...	9 ...	" "
		Mother ..
He. ...	14 ...	" positive

Thus among 13 parents of children giving a positive reaction, 9 were found to give a positive reaction.

It will be noticed that in the above table in the case of the patient Har. a positive reaction was obtained at an interval of fifteen, and in the case of Sc. at an interval of sixteen, years after the birth of a syphilitic child. The period during which a positive reaction may be obtained is known to be extremely variable in the case of the acquired form of the disease. In the congenital form it might be expected that the percentage of positive results would bear a close relation to the age of the patients examined. A grouping of the 330 cases according to age gives the following result :—

	Examined		Positive		Percentage of positive results
(I) Patients aged 10 and under (of these two only were less than 5 years old)	94	...	20	...	21.27
(II) Patients from 11 up to 15 years of age inclusive	142	...	24	...	16.9
(III) Patients from 16 to 20 years of age	66	...	4	...	6.06

Of patients aged from 21 to 30, 24 were examined with 3 positive results. The remaining 8 patients ranged in age from 31 to 44, and all 8 gave a negative reaction.

The above table appears to show that the percentage of positive results diminishes rapidly after the sixteenth year, and that a larger percentage of positive results might be expected from the examination of a series of very young cases. In any case, the average age of the patients investigated must be regarded as an important factor in any estimation of the prevalence of congenital syphilis, and it seems to me possible that the very contradictory results already published may be reconciled by taking the age factor into consideration. Of the 51 cases in which a positive serum reaction was obtained, 7 only showed conclusive evidence of congenital syphilis from a clinical standpoint. In the remaining 44 cases a diagnosis of syphilis rested on the evidence of the serum test.

At the present time it is, I think, generally admitted that a positive result obtained by the Wassermann reaction affords strong evidence of a syphilitic infection, and it is superfluous for me to cite evidence in support of its reliability. The practical value of the test must necessarily be measured by the number of those cases in which the positive serum reaction is the only obtainable sign of a syphilitic infection. In cases where latent syphilis is suspected, a single negative result affords little information, but the value of a positive result has been established beyond doubt. In this connexion great interest has been aroused by the cases reported by Plaut and others, in which the husband or wife of a syphilitic has been shown to give a positive reaction, but has never shown any sign or symptom of the disease. It is even held

that the serum reaction may be the only sign from first to last of a syphilitic infection. Linser [3], quoted by Bruck [1], has examined a series of children of syphilitic patients, and finds that two-thirds give a positive serum reaction, while only one-third show any other sign of infection. Numerous cases are also on record in which it has been demonstrated that the apparently healthy mothers of syphilitic children give a positive reaction, and, conversely, the serum test has been positive in apparently healthy children of syphilitic mothers. The numerous investigations on these lines tend to prove that the evidence of a positive serum reaction may be accepted even in the absence of the usual signs and symptoms of the disease. If, however, it is granted that the 51 cases of this series were the subjects of congenital syphilis, or at least the children of syphilitic parents, it still remains open to question whether there is any evidence of a causal relation between syphilis and idiocy. It might very reasonably be held that the two conditions were unconnected, or that syphilis effected a very remote influence, as a predisposing cause, by in some way impairing the vitality of the parent or offspring. It can also be maintained that a high percentage of congenital syphilis is to be expected among the children of parents whose mental and moral faculties are presumably below the average level.

I had hoped that by a careful examination of those cases which had given a positive result to the serum test it might be possible to detect some symptom or group of symptoms which was common to all. This I failed to do. Very few of the positive cases showed any evidence of a gross lesion in the central nervous system; and this, I think, is quite in accordance with what one might expect, for the gross changes in the brain which are known to be due to congenital syphilis are not compatible, as a rule, with a continuance of life. If a causal relation exists between congenital syphilis and idiocy, the condition which arises may perhaps be classed as parasyphilitic. The absence of the ordinary signs of congenital syphilis in idiocy is closely paralleled in the already authenticated parasyphilitic diseases. It is, of course, notorious that *tabes* and general paralysis commonly occur in patients where the early symptoms of syphilis have been mild or even unnoticed. Among the cases of the juvenile form of general paralysis collected by Mott [5] quite half were found to show no sign of congenital syphilis, but nevertheless to have been born of syphilitic parents and to have brothers and sisters who exhibited the ordinary signs of the disease.

It seems to me reasonable to think that many cases of idiocy should be classed with that form of syphilis which manifests itself alone by a

selective toxic action on the elements of the central nervous system. I do not wish to attach an exaggerated importance to the results of the examination of the serum in one series of cases, but when it can be shown that a considerable percentage of idiots afford evidence of a syphilitic infection, and since it is well known that the virus of syphilis is capable of exercising a selective action on the central nervous system in cases in which there is no other evidence of the disease, I think it is not unreasonable to infer a causal relation between the two conditions.

From the therapeutical standpoint it is hardly, I think, to be expected that any improvement will be obtained by the mercurial treatment of cases of established idiocy, which give a positive Wassermann reaction. On the other hand, it does not seem unreasonable to suggest that the information which can be gained from the Wassermann reaction should be made of use as part of a system of prophylactic treatment. It should not be impracticable or even very difficult to secure a specimen of the blood serum of any woman during pregnancy, in cases in which any suspicion of syphilis might be entertained. If a positive reaction was obtained it can hardly be doubted that the active treatment of the mother, followed later by treatment of the infant, would be the means of averting or modifying the occurrence of symptoms. It has been even suggested by Professor Wassermann that a routine examination should be undertaken of the blood serum of every patient who is admitted to a lying-in hospital. The proposal is the logical result of the belief that a syphilitic infection may exist in an apparently healthy subject, and that a reliable diagnosis can be obtained by the employment of the Wassermann reaction.

In conclusion, I have pleasure in expressing my gratitude to Professor Wassermann for the kind interest which he took in these experiments. My best thanks are also due to the Director and Medical Officers of the Wilhelmstift Asylum in Potsdam for their kindness in assisting me in collecting samples of serum and in the examination of the patients.

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DISCUSSION.

Dr. SHUTTLEWORTH said he had listened with great interest to Dr. Dean's paper, because he thought it likely that the use of the Wassermann reaction, if authenticated in the way that seemed likely, would materially influence the views of the profession as to the relative importance of syphilis in the ætiology of idiocy. Judged merely from the clinical symptoms—i.e., from the stigmata of syphilis, and also from the family history, the result, so far, of inquiries in English Institutions at any rate, had been somewhat disappointing. Years ago, when superintendent of the Royal Albert Asylum, he was engaged with his friend Dr. Fletcher Beach, then medical superintendent at Darenth Asylum, in a case-book investigation of the ætiology of idiocy, and they were surprised to find in how few instances there were signs of syphilis, or a definite history of syphilis, amongst the 2,380 cases scrutinized. He had not been able to refer to the table that day, but he believed there were less than 2 per cent. (1'17) in which they could be quite sure that there was evidence of syphilis, either from clinical signs or from the family history. Their statistics depended, of course, partly on personal scrutiny of the patient, but were also the results of a fairly careful investigation of the family history; not merely accepting the statements made by the friends of the patient at the time of admission, but corrected by personal observation of various members of the family on their successive visits to the asylum, during several years in some of the cases. They were not alone, however, in the paucity of the ascertained results with regard to syphilitic ætiology as a fact of idiocy, because even Sir Jonathan Hutchinson, in a visit to Earlswood Asylum, confessed that he was astonished to find in how few cases there were definite evidences either of inherited syphilis in the child or a syphilitic history. He (Dr. Shuttleworth) had always suspected that there was a great deal more syphilis in the ætiology of idiocy than the statistics available seemed to show. There were various types of idiocy, such as the hydrocephalic, sclerotic, &c., in which there might be fair presumption of a syphilitic causation, though absolute proof could not be brought forward. He thought examination by the Wassermann reaction would probably enable physicians to attach a much larger importance to the share of syphilis in the causation of idiocy, and indeed of mental defect generally. One might also extend that examination not merely to idiots and imbeciles found in the idiot asylums of the country, but to many of the mentally defective children who form such a considerable proportion—nearly 1 per cent.—of the school population. One reason why in institutions one did not find a larger proportion of syphilitic causation was the fact that in bad cases of inherited syphilis the subjects were usually dead before the age at which admission was customary at institutions—i.e., 7 years of age. Moreover, in some cases the mental breakdown due to inherited syphilis did not occur until after the period of the second dentition (i.e., in cases of juvenile general paralysis), and these one saw in an early stage rather in school children than in idiot institutions. Such children

might present no overt signs of syphilis on their persons, and in many it might be impossible to obtain a syphilitic family history. These often got through the first three or four standards at school and broke down in the fifth or sixth, and then passed into a condition of progressive dementia which, of late years, had been called "juvenile general paralysis." In regard to the latter condition, Dr. Mott, in his investigations, found a comparatively small percentage of cases in which there were syphilitic stigmata to be seen on the patients themselves. But on careful examination of members of the family, brothers and sisters, &c., he was enabled to attribute as many as 80 per cent. of his forty cases of juvenile general paralysis to inherited syphilis.

Dr. HEAD asked particularly with regard to the case where there was a positive reaction in the cerebrospinal fluid. He did so because the triple reaction was now carried out frequently in cases of cerebrospinal syphilis or parasyphilis—the reaction of the blood, of the cerebrospinal fluid, and the leucocytic count, with possibly the globulin test. If a positive reaction was obtained in all, it showed the affection to be parasyphilitic, not syphilitic. Mott had expressed this view strongly in his recent work. In the present series there seemed to be a positive reaction in the blood and in the cerebrospinal fluid in one case only; one would not expect this to occur in any case unless the patient had juvenile general paralysis or juvenile tabes.

The CHAIRMAN (Dr. J. A. Ormerod), commenting on Dr. Head's remark, said if, as Dr. Dean hinted, the cases were not those of gross syphilitic disease, but idiocy due to something parasyphilitic, then the surprising thing was that Wassermann's reaction was not more often positive in the cerebrospinal fluid.

Dr. DEAN, in reply, said that the youngest patient of the series was aged $3\frac{1}{2}$. In infants and young children, when it had not been possible to get blood from the vein, he had tried various kinds of cupping apparatus, but with very little success. In such cases it was probably easier to prick the finger or ear and collect the blood in an ordinary "Widal" tube. Very small samples of blood were difficult and unsatisfactory to work with. The case mentioned by Dr. Head had no special features from a clinical standpoint; it was not a case of the juvenile form of a general paralysis. Specimens of blood from the father and mother of this patient were examined and gave a negative reaction. He instanced the cases of two brothers who both gave a positive reaction without other sign of syphilis. Considering that the percentage of positive results amounted to 15 per cent., the occurrence of a positive result in the two brothers was probably not a coincidence, and might be regarded as evidence of the reliability of the reaction. He examined fourteen cases of marked hydrocephalus, and only four gave a positive reaction. He looked carefully for signs of hydrocephalus among the positive cases, and, in addition to the four he mentioned, which were very obvious, there were three others which showed slight hydrocephalus. But ten obvious cases of hydrocephalus gave a negative reaction.

Secondary Growths affecting Spinal Roots.

By E. FARQUHAR BUZZARD, M.D.

WITH PATHOLOGICAL REPORT

By C. M. HINDS HOWELL, M.D.

A MAN, aged 40, was admitted into the National Hospital, Queen Square, on April 8, 1910, with the following history: In October, 1909, he began to suffer dull, aching, intermittent pain in the right forearm and noticed numbness in the fingers of the right hand. By December the pain involved the whole of the right arm and the left leg below the knee, and by January had spread to the left side of the head and neck. In March similar pain attacked the left arm, the right leg, and the right side of the head and neck, and by April he was unable to walk on account of paresis of both legs. At the same time he developed some difficulty in swallowing both liquids and solids, some embarrassment of respiration, and some slight indistinctness in articulation.

On admission he appeared gravely ill. He had a rapid but regular small pulse of 132, without any obvious enlargement of the heart. His temperature was raised, and he had signs of consolidation at the base of the left lung. Nothing abnormal was detected in the abdomen. The function of the cranial nerves was natural except for (1) slight weakness of the right side of the face; (2) some difficulty in swallowing, described as a feeling of not knowing what to do with food when it reached the pharynx; (3) tongue protruded slightly to right; and (4) weakness of the sterno-mastoids and trapezii on both sides. There was weakness of both arms, more marked on the left side and especially affecting the extensors of the left wrist. Wasting was apparent in the ulnar group of muscles and in the intrinsic muscles of each hand. The muscles of the trunk were generally weak. Both legs were weak, the flexors of the ankles and thighs especially so, and there was wasting of the muscles of the feet. No defect of sensibility on testing. Probably some increased tenderness of muscles on deep pressure; this was particularly noticeable in the left upper extremity. Pains and numbness were complained of, as already stated. All the tendon jerks were lost except the right wrist-jerk. The abdominal reflexes were not obtained. The plantar responses were of an indefinite flexor type. The sphincter control was perfectly normal.

The patient died rather suddenly before he had been in hospital for three days.

Dr. Buzzard thought the case was worth recording for two reasons. In the first place, the clinical history and examination gave no definite clue to the real disease as discovered after death. In the second place, the case served to emphasize the necessity for microscopical examination of the nervous system before any statement could be made about the presence or absence of gross disease. On the post-mortem table nothing was to be seen in the spinal meninges or spinal roots to make one suspect what was found in the sections. In the same way the presence of meningitis ought never to be denied without careful microscopical investigation.

PATHOLOGICAL REPORT.

Post-mortem five hours after death. Body well nourished, much post-mortem lividity in dependent parts.

Thorax: Pleura natural. Lungs—Collapse of left lower lobe; on section, there was marked œdema of lungs in remaining areas. Microscopical examination showed the collapsed area to be the seat of a lobar pneumonia in an early stage of hepatization. The lungs were free from growth. Pericardium—Recent mediastinitis, with adhesions to outer layer of pericardium. The latter was distended with fluid; on cutting into it about a pint of dark, blood-stained fluid escaped. Intense fibrinous pericarditis was present. Heart—A hard nodule was felt within the heart, which proved to be a sarcoma lying chiefly in the right auriculo-ventricular septum, with some smaller nodules under the endocardium in the right auricle. Liver—Enlarged and hard; surface smooth, but scattered over the surface, particularly towards the anterior border of the liver, were a number of small, round, opaque areas, mostly of the size of a split pea; these were just appreciable on palpation with the finger. On section, the liver showed early chronic venous engorgement, with some increase in fibrous tissue. Microscopically the areas referred to proved to be small foci of sarcomatous cells.

Kidneys and suprarenals: Both suprarenals were found to be the seat of new growth, which had retained the general shape of the capsule but had caused considerable enlargement of these organs. The growths were smooth, pale, and hard; the right growth and capsule weighed $1\frac{1}{2}$ oz., the left a little less. The general character of both growths was the same. The kidneys were of good size; the capsule stripped readily. The growth in the suprarenals had not infiltrated the kidney directly,

but in the upper part of both kidneys, particularly the right, and situated in the cortex of the gland, were several hard, white, discrete nodules of growth.

The abdominal lymphatic system was carefully examined, but no enlarged glands were found, nor any further macroscopic evidence of growth. One or two lymphatic glands were examined microscopically, with negative result. The abdominal contents were otherwise normal. Microscopically, all the growths in the viscera presented similar appearances, the tumour in each instance being a round-cell sarcoma.

Brain and spinal cord: Bones of skull and the vertebræ appeared normal. The meninges of brain and cord macroscopically showed no

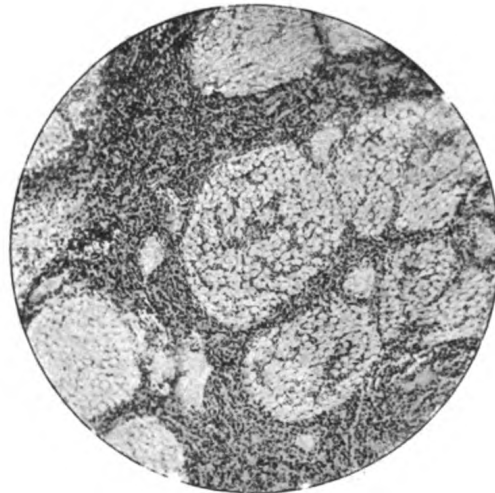


FIG. 1.

Photomicrograph of spinal nerve-root showing intense sarcomatous infiltration.
(High power.)

evidence of growth, but the spinal vessels were very much engorged, chiefly in the lower part of the cord, and the spinal nerve roots seemed somewhat swollen, this being particularly noticeable in the cauda equina. The spinal cord itself appeared somewhat swollen and œdematous, but no gross macroscopic lesions could be detected. The brain appeared normal macroscopically; the cranial nerves, however, especially the third pair and the ninth, tenth, and eleventh, appeared somewhat swollen and œdematous. No secondary growths were found on making sections of the brain.

Sections were taken from the cerebral cortex, pons, medulla, and spinal cord, and examined by various methods.

Hæmotoxylin and Van Gieson: Throughout the whole spinal cord, and extending up to the medulla, there was evidence of sarcomatous infiltration, which had affected the meninges and the spinal roots; this infiltration was as marked in the cervical as in the lumbar segments. The most striking feature which the sections disclosed was the marked disproportion between the infiltration in the roots and the meninges. Whereas

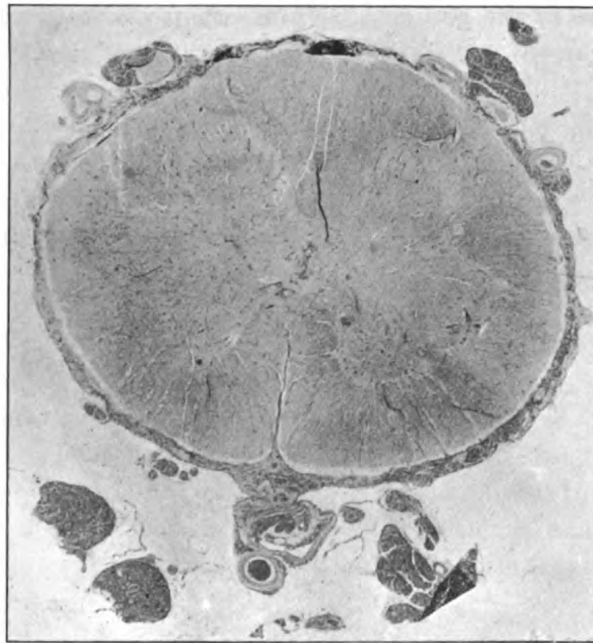


FIG. 2.

Photomicrograph of a sacral segment of spinal cord. Note thickened meninges and sarcomatous infiltration round anterior spinal artery. (Low power.)

both anterior and posterior roots were the seat of a very intense infiltration, the meninges were affected comparatively slightly, though around the anterior and posterior spinal arteries the sarcoma cells were to be seen in great numbers; but even here there was less growth than in the spinal roots. There had been no direct infiltration of the spinal cord, but sarcoma cells could be seen in a few instances in the perivascular spaces of the intra-medullary spinal vessels, particularly in the lumbar segments

of the cord. Examination of the higher parts of the brain-stem showed a slight sarcomatous infiltration in the lepto-meninges as high as the mid-brain, in addition to which, throughout the whole lepto-meninges, there had been a slight inflammatory reaction. A very well marked nodule of growth was seen in one of the sections through the medulla, attached to the accessory vagal root.

Nissl method: This showed very clearly definite chromatolysis in the cells of the ventral horns. Various stages could be seen, from a slight perinuclear change to advanced chromatolytic degeneration.

Marchi method: (a) Spinal cord—Only slight degeneration of a few scattered fibres in the posterior columns was to be seen: Spinal roots—

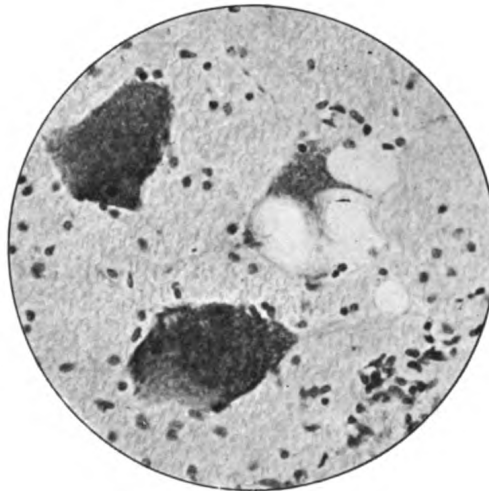


FIG. 3.

Photomicrograph of ventral horn cells showing marked chromatolytic changes.
(High power.)

There was some degeneration in the ventral roots, but not very pronounced. (b) Medulla—The fasciculus solitarius showed fairly well marked degenerative change, as did some of the intra-medullary fibres of the twelfth and vagal accessory. (c) The vagi, phrenic, median, and external popliteal nerves were examined, but very little degeneration was found in any of the fibres. (d) Muscles—The thenar and tibialis anticus muscles showed groups of individual fibres, in which early fatty changes in the form of a fine granular degeneration could be easily distinguished.

Transverse and longitudinal sections were also made of the same muscles and stained by hæmatoxylin and Van Gieson. The muscle fibres showed definite changes in a few of the muscular fasciculi: these consisted of a swollen appearance in some of the fibres, whilst others were much diminished in size and atrophic. There was no considerable proliferation of nuclei of the sarcolemma.

The condition of the muscle-spindles is worthy of mention. In the muscles examined only a few seemed definitely normal; the majority showed an apparent distension of the connective-tissue sheath, with some coagulable material, and in a few instances the small muscle fibres within the spindle showed vacuolation, which was not to be seen in the muscle fibres throughout the rest of the section. Central nuclei were also to be seen in some of the intra-fusal fibres.

The result of the microscopical examination of the central nervous system would seem to show that the nerve-roots had been first invaded through the intervertebral foramina, and that subsequently the meninges had become involved. The sarcomatous deposit had gradually tracked up the spinal cord to the mid-brain, where the meningeal invasion was slightest. This conclusion is justified by comparing the intense infiltration of the roots with the relatively slight degree with which the meninges had been involved. In two other cases that I have examined recently, in which deposits had occurred in the meninges secondary to cerebellar growth, the roots had remained relatively free and were to be seen passing out through the meningeal tumour without being infiltrated by it. Apparently the perineurium had offered considerable resistance to the growth, and had thereby prevented infiltration of the nerve fibres.

DISCUSSION.

The CHAIRMAN (Dr. J. A. Ormerod) said members would recall that many years ago one such case was published by Dr. Hughes Bennett, and in that case the posterior roots were particularly involved, and the clinical aspects of the case were almost exactly like those of tabes.

Dr. F. E. BATTEN said there were one or two points which he thought of interest in the case. First, with regard to the muscular condition which Dr. Howell showed, in which there was fatty degeneration of certain fibres; that was met with under many conditions. In patients who had had considerable wasting in almost any nervous disease the same condition would be seen. It was met with in paralysis agitans and certain myelopathic conditions, in progressive muscular atrophy and tabes. Secondly, with regard to the

muscle spindles, in the section shown by Dr. Howell, it was very difficult to express an opinion on a section through any one portion of a muscle spindle. It was well known that a section through the equatorial region of a muscle spindle differed greatly from a section cut through one of the poles. The muscle fibres at the equatorial region lost their striation at a certain point; they contained curious big cells, and the nuclei became central in the muscle fibres. The space which Dr. Howell described with a homogeneous material he would have thought was compatible with the normal lymphoid space which nearly always existed at the equatorial region. He could not say there was anything definitely abnormal in the section shown by Dr. Howell, but it was impossible to express a definite opinion without seeing serial sections, and it was difficult to get such a series of a muscle spindle.

Dr. HOWELL replied that he was glad to hear Dr. Batten's opinion about the muscle spindles, but in each of the muscle sections examined there were many spindles which presented the appearances described, and they could scarcely all have been cut through the equatorial region. He would, however, try to get serial sections.

Acute Cerebral Softening, ? due to Venous Thrombosis.

By WILFRED HARRIS, M.D., and BERNARD H. SPILSBURY, M.D.

W. T., A TRAVELLER, aged 28, was quite well until the evening of April 21 last, when he complained of slight headache. He went to bed as usual, and at 3 a.m. his wife awoke and found him groaning and vomiting. He was then seized with left-sided convulsions, which lasted for two hours, followed by unconsciousness. Next morning he was found to be comatose, and a slight left-sided convulsion again seen. He was then sent up to St. Mary's Hospital for admission under one of us, when he was found to be quite comatose, with flaccid left hemiplegia. There was no divergence of the eyes or conjugate deviation. Pupils equal, medium and equal, no reaction to light, disks normal, except for some retinal arteriosclerosis. Right knee-jerk present, left absent. Right plantar flexor, left extensor. Pulse, 120; temperature, 102.4° F.; respiration, 32. Lumbar puncture was then done by one of us, and 10 c.c. of clear fluid obtained, under low pressure. This was followed by rapid breathing and a pulse-rate of 60. At 5 p.m. the pulse recovered, 128; respiration, 55, becoming more irregular. Irregular movements of the right arm and leg were occasionally present, but no

movement on the left side. The left arm later became stiff. At 10 p.m. the temperature rose to 104.4° F., and death occurred.

At the autopsy, held sixteen hours later, the surface of the brain appeared normal, and careful dissection revealed no embolism or thrombus in any superficial artery or vein. On horizontal section of the brain, in the right hemisphere there was found an extensive area of acute softening involving the whole area of the section with the exception of the occipital lobe and the anterior half of the frontal lobe. This area of softening extended into the corpus callosum just to the left of the middle line, the white matter appearing soft and translucent, and containing numerous punctate hæmorrhages. The grey matter was normal. There was no evidence of meningitis, either macroscopically or microscopically. No thrombus was seen in the veins of Galen. The arteries at the base were normal, including the whole of the internal carotid in its bony canal. Microscopically, large numbers of recent minute hæmorrhages were seen, and also hæmorrhages into the perivascular lymph spaces of some of the arteries. There was slight polynuclear infiltration of the white matter. In the lungs patchy broncho-pneumonia was found, the inflamed areas containing numerous Gram-positive streptococci and diplococci resembling pneumococci. Heart, liver, pancreas, and kidneys normal.

Remarks.—The diagnosis of the cause of an acute hemiplegia is often extremely difficult. In this case, a young man, aged 28, neither the condition of the radial artery nor of the heart gave any reason to suspect hæmorrhage, thrombosis, or embolism. The sugar in the urine was considered to be a shock glycosuria, due to the sudden cerebral lesion. The flaccid left hemiplegia, with extensor plantar reflex, pointed to the lesion being probably in the right cerebral hemisphere, but there was no conjugate deviation of the eyes and head, or dilatation of one pupil, as is frequently found in such conditions. Hæmorrhage into a growth was considered, but there was no optic neuritis, and the history of previous perfect health appeared to exclude this. The onset was much more sudden than is usual in acute encephalitis, and the absence of microscopical evidence of patchy excessive leucocytic infiltration are against this diagnosis. In our opinion the condition of acute softening found, with the numerous minute hæmorrhages, is best explained on the theory of venous thrombosis affecting afferent veins leading from the subjacent white matter to the surface, though no actual evidence of this condition was found, the larger veins on the surface and the veins of Galen, as well as the arteries, being all normal.

DISCUSSION.

Dr. F. E. BATTEN asked whether the urine had been examined, as some of the acute cases of hæmorrhage with sudden onset had been attended with sugar in the urine. Whether that was cause or effect he did not know.

Dr. FARQUHAR BUZZARD doubted whether it was possible to exclude encephalitis. There was a very short interval between the onset of the symptoms and death, and in acute virulent injections one might get very little more in the way of cellular reaction than was found in this case. In support of an effective origin there was the fact that petechial hæmorrhages occurred elsewhere. He did not think encephalitis could be excluded.

Dr. TREVELYAN said he thought it might be a case of acute hæmorrhagic encephalitis, such as had been described by Oppenheim. During an epidemic of influenza in Leeds there was a case of unilateral convulsions, followed by paralysis. In the brain, chiefly in the cortex, there were foci of disease similar to those which had been described here.

Dr. HARRIS, in reply, said it was impossible to prove that there was polio-encephalitis, or, what he believed to be the same, acute encephalitis. But the question occurred to both of them, clinically and pathologically. From what he knew of the anatomy of acute encephalitis, he did not believe the section bore out that idea. It looked as if there was acute softening following a large thrombus. It attacked the white matter and the central portion, and extended into the corpus callosum, without involving the cortex anywhere. His acquaintance with the morbid anatomy of such acute cases of encephalitis was slight, but he thought they always involved some part of the grey matter. He shared Dr. Spilsbury's view that this case evidenced obstruction somewhere, although it was not discovered.

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SESSION 1909-10

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1910

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Obstetrical and Gynæcological Section.

October 14, 1909.

Dr. H. MACNAUGHTON-JONES, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

Pain associated with Disorders of the Female Genital Organs.

MY first duty, as it is my natural desire, on taking this chair is to thank the Fellows of the Section for placing me in it; and in doing this I am not forgetful that it is primarily to my old associates of the British Gynæcological Society that I am indebted for this great honour, when they nominated me, in accordance with their compact with the Obstetrical Society, as the successor of the first President, Dr. Herbert Spencer. His masterly guidance of this Section, during the first two years of its existence, has created a presidential standard of such excellence that it places a most difficult task before anyone who has to follow him. I can here truthfully, and without affectation, say how fully I realize the responsibility attaching to the position I have accepted, and my own deficiencies in filling it. It is a somewhat unique one from the fact that I, who have never been attached to an English hospital or school, should find myself thus honoured—a fact to be explained only by the generous courtesy of my English brethren. I hope for the same generous consideration during my year of office, trusting that at its conclusion I may not have proved altogether unworthy of this mark of confidence.

When I learnt that a short introductory address would be expected of me on my assumption of office, I felt a difficulty in selecting a topic sufficiently interesting to all that might be briefly dealt with in a satisfactory manner, and yet leave some impression on my listeners. With this dual object in view I have selected for my theme the subject

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of pain, applying my remarks, specially and particularly, to that which is associated with disorders of the female genital organs.

There are many aspects from which we may view the existence of pain. Primarily we may classify these under two heads—the metaphysical and the physical. Much of what is purely visionary and speculative has been written with regard to the former.

A distinguished personal friend of mine, to whom in the past I was indebted for many kindnesses, and whose ideals were not merely mythical will-o'-the-wisps, mental *muscæ volitantes*, but actual living potentialities, objective realities to be grasped and incorporated with the individual life—the late James Hinton, philosopher and otologist—wrote a short but remarkable brochure entitled “The Mystery of Pain.” In it the view of pain from the psychical standpoint is discussed. “We have,” as he says, “to accept pain as a fact, existing as a deep necessity, having its root in the essential order of the world,” as I have myself expressed it—

And if each soul its cup must fill,
And deep the dregs must drain,
'Tis part of the Eternal will—
The Mystery of Pain.

If this be true, and I believe it is, then, recognizing the universality of Evolution's operations in nature, and realizing that from the nebulous spiral seen in the genesis and growth of a world to a spirochæte, there appears to nature's eye to be a unification of design and scheme, “the groaning of the whole creation” occupies its place in the working out and completion of a design that embraces alike the star and the micro-organism. Certain it is that pleasure and pain are very closely allied—

For Pain sits ever tight on Pleasure's back,
With lifted spur, a shadow on her track ;

and one of the greatest of retrospective pleasures is the remembrance of the endurance and conquest of pain. Does not this anticipated sequence of the endurance of pain explain much of human effort, even when that effort has no possible reward save the acknowledgment by his fellows of the man's triumph over obstacles that involved in their conquest privation and suffering? Witness the late achievement of the asserted conquest of the North Pole. Pleasure and pain are a part of that law of action and reaction to which we all, rich and poor alike, wise man or fool, have to submit.

First, I would say a few words viewing pain from the negative aspect. It is remarkable how women generally bear pain, though they may vary greatly in their fortitude and their sensitiveness to it. Nature's decree that in great suffering the woman must bear her offspring would appear to have a corollary in the increased capacity of the sex to endure. In my experience, women bear pain much better than men, and it is sometimes the woman from whom we least expect it who is most patient and shows the greatest courage when she is put to the test. It may be that it is this tacit recognition by men of woman's power of endurance, and the man's familiarity with her resignation under the painful burden Nature has placed on her in the discharge of her sexual functions—in many cases amounting to years of recurring monthly suffering—that frequently leads him to slight, if not to altogether ignore, her complainings, and even those to whom she might more reasonably look for sympathy are often too inclined to ascribe her complaint to "temperament" and "nervousness."

"Neurosis," "neurasthenia," and "hysteria" are terms that are convenient excuses when patience and trouble are alike demanded in a reasonable search after the true cause of the woman's complaint. I know of no more dangerous terms in the gynæcological vocabulary than these. They are apt to lead the young or unwary practitioner into a diagnostic cul-de-sac, missing in his stumble therein the track to some serious pathological condition that this delusive impression has prevented his reaching. And, indeed, I have heard it whispered that some gynæcologists of the gentler sex, doubtless rendered a little callous through their education by the harder, are apt to fall victims to the same error.

Pain, whether it have a physical or a mental origin, is an entity that has to be fought, and that which is subjective in character is as real to the sufferer as that which is objective. I do not in the least infer that there are not many morbidly introspective women who suffer from delusive impulses and pains that are truly imaginary, and who are, in the strict acceptance of the term, neurotic. Thus, out of the catamenial crisis and during the nerve storms of the climacteric, we have actual hysteria and neurasthenia leading up to, and oftentimes across the borderland of, disorder of mentalization. We all know the patient who, if you do not interrupt her, will pass in review every part of the body from scalp to toe, to which she pathetically points as the seat of pain. None the less has she to be treated for this.

But from this allusion to capacity to endure on the one hand and

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marked and exaggerated sensitiveness or wholly subjective symptoms on the other, I come to the important fact that the gravest pathological conditions in the genitalia are often not attended by much actual pain, and we are amazed on examination to find an amount of disease present which we cannot conceive progressing without anything more being felt than some "discomfort" and "slight suffering." We are surprised by the assurance that there has been "no pain to speak of." This is so when even the bowel and bladder are mechanically affected by pressure.

I show an enormous and rapidly growing scirrhus carcinoma of one ovary, and fibromyoma of the other, removed from a patient who for some time believed she was pregnant, and was astonished when she was told she had a tumour; she had suffered so little. There is also a large double pyosalpinx with uterus, in which the most extensive adhesions of uterus and adnexa are seen. The patient persisted that it was only the trouble of the bladder, which in this instance was enormously distended, that forced her to seek advice.

I have a patient at the present moment who has recovered from a most severe operation—a double pyosalpinx with extensive bowel adhesions. She cannot understand how anything serious could have been the matter with her; the day before she came into the home she was "quite happy making butter." But from this same patient we may learn another lesson. Some years since she complained of persistent pain in the right side, and as this was complicated with dysmenorrhœa it was not taken much notice of, and examination was put off. Then she had a chill, from which she dated the aggravation of her symptoms. Had the cause of the pain at the time been inquired into, the graver troubles might have been averted.

In connexion with this early attention to pain, it is worthy of remembrance that it not infrequently happens that pain is felt much more acutely in the incipient stages of a disease of the genitalia than in its later and more complete developments. The primary effects of invasion of healthy structures and the sudden interference with functional activity, the consequences of commencing encroachment on adjacent organs, and the more acute symptoms of the onset of inflammation, explain this. It is not so much of the present suffering that the woman often complains as the decline in her health generally from secondary and collateral results. Close inquiry may reveal the fact that years have elapsed since pain was first experienced; delicacy on the woman's part, dislike or fear of interference, and the apathy of friends have contributed to the neglect of nature's demand for attention at the

critical time when the proverbial "stitch in time" would in reality save the need for many a suture and ligature in the future.

There is nothing uncommon in discovering inoperable cervical carcinoma with invaded parametrium, when, though there has been hæmorrhage, little or no pain has been experienced. The hæmorrhage relieves the pain and washes away the malodorous discharge. There is here a giant myoma which caused no symptoms, save some inconvenience, until it pressed on the diaphragm and caused dyspnœa and tachycardia. There is also a myoma with a large telangiectatic cavity which was full of blood, that never caused any pain, and the patient first drew attention to it by saying she had "a lump in her side." I show a large myomatous uterus in which a necrobiotic mass, surrounded by calcareous degeneration, is seen in one of the tumours. So little trouble did it give that a very eminent gynæcologist expressed his opinion that operation was not a necessity. So in tubercle, papilloma, and dermoids of the ovary; these and other pathological invasions occur, and but trifling pain is felt almost up to the time when operative interference is called for. An ectopic gestation will cure a persistent dysmenorrhœa, and its presence is often only suspected by the rupture of the sac.

A few years since I removed a hydatid cyst of the liver from an unmarried woman, drawing off some 5 pints of echinococcus fluid. Until a few weeks previously she had never complained of pain, and her condition at first closely simulated that of muscular rheumatism. She made a complete and permanent recovery. All this, you will say, is a common experience. It, however, establishes the first point I wish to emphasize with regard to the clinical value of the symptom—namely, that its absence may be consistent with the presence of the gravest pelvic lesions.

On the other hand, we see the reverse of this when the pain is out of all proportion to the clinical signs and the gross pathological changes. We have a good example in the instance of sclerotic and cirrhotic ovaries. Vaginal examination reveals nothing save a somewhat enlarged, if hard and sensitive, ovary, and yet the dysmenorrhœa is such as to cause delirium and bring the sufferer, barely recovering from one attack when another supervenes, to the verge of insanity. This is true "dysootocia," as Robert Barnes termed it. The condition is greatly aggravated when there is an associated menorrhagia. The cause of the pain we see revealed in the complete fibrotic ovary with obliterated follicles and vessels and degenerative cystic formations. I lay some good examples of such ovaries before you, with stained sections

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illustrating the histological changes. The pain in these cases is suprapubic, radiating all round the pelvis and down the thighs, much as it occurs in stenosis of the isthmus uteri, and its attendant congestive dysmenorrhœa.

Pain has to be regarded from its clinical, pathological, and therapeutic bearings on diagnosis, prognosis, and treatment respectively. And first touching its assistance in diagnosis. This has to be considered under three heads: First, that which is felt when the person is at rest or during the night, disturbing sleep; secondly, that which is aggravated or only experienced when moving about; and, lastly, that which is elicited by palpation, for exquisitely sensitive parts may thus be discovered, though otherwise not painful. Further, in relation to each of these subdivisions, we have to consider the character and the situation of the pain.

It would obviously be impossible in a short address to go into all these parts in detail. I must therefore generalize, bringing into prominence some that I consider most important. Speaking broadly, it may be taken that the pain which is felt when at rest and prevents sleep is of an active, inflammatory source, and if referred to the "sides"—that is, the iliac regions—is usually due to an ovarian or tubal affection. Abdominal and rectal palpation will confirm this, and the sensitiveness of a prolapsed ovary or inflamed tube is characteristic in its sharpness.

The tenderness of a prolapsed ovary may only be manifest through coitus, and the severe dyspareunia that attends on an exquisitely sensitive introitus and hymen draws attention to some vaginal or hymeneal trouble not otherwise complained of.

Pressure on the cervix made in the direction opposite to that of the affected adnexa, by pushing the fundus on to these, increases the pain. This latter is not necessarily felt in the affected side. An examination discloses a swelling in the other fornix. This may be due to transposition or various adhesions which drag on the healthy adnexa.

A constant though shifting pain, sometimes of a paroxysmal character, in the right iliac and inguinal regions may have a quadruple origin—kidney, ureter, appendix, or adnexa. There is only a slight non-sensitive swelling to be found in the vaginal fornix, or we may detect none, yet by deep abdominal pressure we elicit pain in the classical spot of appendical trouble. There is a complicating history of constipation and old abortive attacks of appendicitis. In such a case there may be both appendical and adnexal complications. Here radiography

is an important aid to differential diagnosis, which latter is often very difficult, though a complete urinary analysis and the collateral symptoms will assist us. It is one of those conditions in which the old surgical rule with regard to hernia is applicable—"when in doubt, operate." Only when we expose the parts do we ascertain the cause or causes of the pain. I show an appendix matted to the adnexa, with two large concretions, one still in the appendix. Here a differential diagnosis would be impossible. When there has been a previous cœliotomy we may light on nothing save an appendical, omental, or pelvic adhesion.

Pain in some distant part due to pelvic trouble is not uncommon: witness that in the intercostal or scapular region simulating pleurisy which has been noticed in ectopic gestation, an instance of which I brought before this Section last session. Headache, migraine, and epigastric pains are often associated with affections of the genitalia. Independently of its accompanying backache, a retroverted uterus is responsible for various painful reflexes and neuroses.

We have to be careful lest, having discovered some uterine affection, we ascribe the head symptoms to this, when they are rather due to astigmatic refraction, while the gastric ones are the result of dyspepsia, or possibly some serious gastric derangement. A solid tumour of the ovary pressing on the sacral nerves will cause pain in the hip and lameness in walking. I show such a tumour which was pressed down in the pelvis by a myoma, and the symptoms closely simulated those of morbus coxarum. The constant boring, burning pain felt in the back, and radiating round the pelvic brim, is very characteristic of cancer of the uterus. The passage of pelvic into general peritonitis has an unmistakable indication in the alteration in the character and extent of the sensitiveness and suffering.

In regard to the bladder and micturition, pain before the act is more frequently due to an exaggeration of the normal uterine angle, an ante-flexion, a myoma in the anterior uterine wall, or some cystic irritation and inflammation. Pain during micturition has usually a urethral cause, either in the nature of the urine or a urethritis from a vaginal source, a growth, a contraction, or pressure from a uterine tumour. That felt after the bladder is empty is more likely to be caused by calculus, cystitis, or urethral inflammation.

There is that most difficult and resistive symptom to treat—pain with frequency of micturition, and lasting for some time after the urine is passed, due to circumscribed inflammation in the trigone, with injection of the mucous membrane in the neighbourhood of the ureteral

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orifices, and perhaps a little pouting of one or both of these. Here Kelly's or other method of cystoscopy and exploration of the ureters and kidneys, assisted by Röntgen examination, may be necessary to save us from overlooking a ureteral or renal cause, though the situation and direction of the pain, antecedent to the emptying of the bladder, will arouse our suspicion of this. The source of the pain, when there is a colitis associated with an adnexal affection (especially if it partake of the nervous type), is often difficult to differentiate. The removal of the adnexa may not bring relief from the pain. In one such case I reopened the abdomen and freed adhesions which had formed from a previous cœliotomy, binding the uterus to the sigmoid, and removed the right cystic ovary, but without affecting the pain.

A late distinguished gynecologist removed an ovary and left the uterine portion of the Fallopian tube. After some time pain returned in the old situation; nothing relieved it. Some years subsequently I opened the abdomen, and found the uterine portion of the tube twisted on itself and firmly adherent to the uterus, which was drawn over by adhesions to the rectum. Complete and permanent relief followed rectification of these secondary results.

Appendical adhesions are not uncommon. As a cause of pain after cœliotomy, Krönig and others make a rule of removing the appendix in every cœliotomy where the delay is not riskful.

A good instance of the influence of the situation of the disease on the presence or intensity of the pain may be seen in sarcoma of the uterus. I have known extensive sarcoma which was entirely confined to the mucous membrane and attended by comparatively little pain, whereas that which is interstitial and invades the parenchyma of the uterus (as noticed by A. R. Simpson) causes considerable and often paroxysmal attacks of pain, sometimes of an expulsive character.

The pain of coccygodynia is characteristic, and, apart from fracture or luxation, has usually a nervous origin. It has its counterpart in the obstinate neurosis, often difficult to relieve, in the lower end of the rectum in the vicinity of the anus, and which is not due to hæmorrhoids.

In estimating the prognostic value of post-operative pain we must apportion this in its relation to the other symptoms of bowel, temperature, and pulse. I think Lawson Tait was right when he regarded that pain which comes on early and is referred to the epigastric zone after cœliotomy as the most serious. It is not infrequently the precursor of septic invasion. This epigastric pain is interesting in the light of our knowledge of the dangerous gravitation that may occur of serum or pus

to the diaphragmatic region. This is just one of the serious drawbacks to the Trendelenburg position in cases where there are pus collections in the pelvis. Pain in this zone of danger is of special significance, especially after a prolonged operation when the body is partly inverted. It indicates also the adoption of the Fowler position, either partial, with the thorax raised, or with the trunk more completely propped up in practically the sitting posture. It is also a warning to adopt at once that most valuable of all recent antiseptic measures—namely, the proctoclysis of J. B. Murphy. This continuous saline rectal irrigation, by his very simple method, converts the peritoneum into a vast secreting rather than an absorbing membrane, and, as he says, “it completes in the very safest manner its own toilet.” It may at times be needful to assist such proctoclysis by suprapubic drainage. Its value in other than obstetric and gynæcological complications as a treatment of peritonitis has been amply demonstrated.

Another practical point in connexion with these secondary operations, especially if chloroform has been administered in the first instance, is the kind of anæsthetic which should be given. From the researches of Harold Stiles and Beesley it is clear that ether is the anæsthetic that should be administered; chloroform, through its causing the formation of acetone, being specially dangerous where acute suppuration is threatening. Indeed, it is a question if we may not have, where toxæmia is feared, to abandon our routine use of chloroform and exercise greater caution in selecting our anæsthetic according to the nature of the case. Very important is it to note that Besredka, in his experiments on anaphylaxis, showed that ether administered before, during, and after the administration of the second dose of the anaphylactic to animals neutralizes the toxic action of the sensibilisin generated by inoculation, rendering them refractory. He states that the administration of chloride of calcium has the same antitoxic effect.

The boring, sacral pain, more especially in operations done by the vaginal route, also brings with it a warning note. Speaking broadly, I think the almost universal view of gynæcological surgeons is to regard morphia as a *dernier ressort* in post-operative pain, and this for sound clinical reasons. Yet I am convinced that it is dangerous to make this a hard-and-fast rule, for there are exceptions when the combination of morphia and atropine is invaluable as a calmative in securing sleep and controlling anæsthetic vomiting. The scopolamine-morphia-and-chloroform method of anæsthesia certainly lessens post-operative pain and sickness.

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I have said nothing of pain as Nature's danger signal, attention to which in the first instance would anticipate and enable us to prevent the advance of serious, if not fatal, complications later on. The sound rule is never to despise, and always to search for, the source of pain, even where it may seem to be of a trivial nature.

Neither have I touched on the indications that are afforded by this analytical and differential study of pain in our choice of therapeutical measures, whether operative or other. The modern armamentarium of the gynæcologist, outside resort to operation, is a large and comprehensive one, whether pharmaceutical or hygienic. Nor should we altogether despise Hilton's sound aphoristic dictum as to the enforcement of rest, with its more modern accessory aids in diet, electro-therapy, and massage. While, however, I am no advocate for the neglect of any therapeutical means we may safely adopt before resort to operation, I have a conviction, seeing the safeguards with which we surround all modern operative interference, that there is, on the whole, greater danger to life in postponement than in early operative relief.

Dr. CHAMPNEYS proposed that the thanks of the Section be given to the President for his eloquent address, and that he be asked to allow it to be printed.

Mr. W. D. SPANTON said he seconded the resolution of a vote of thanks to the President with the utmost pleasure. They had listened to a most interesting and suggestive address, the outcome of the ripe experience of Dr. Macnaughton-Jones, graced with that charm which always characterized him. Philosophers have told us there is no such thing as pain, but the President had clearly shown how pain may exist without any tangible physical change, while tissue changes of the gravest character are found where pain has never been felt. Mr. Spanton thought it well that our attention should be directed to these well-known facts in the delightful manner it had been that evening.

**Case of Placenta Prævia in which it was deemed advisable
to perform Cæsarean Section.**

By J. M. MUNRO KERR, M.D.

THE attitude of obstetricians towards Cæsarean section in certain cases of placenta prævia has altered considerably in recent years, especially in America and Germany. I have been following this movement with great interest, and have for several years thought that this radical treatment was quite justifiable in certain well-chosen cases. I had not, however, seen such a case until the other day.

The following is a history of the case :—Mrs. P., aged 28, was seen by me, along with her medical attendant (Dr. McEwen, Helensburgh), one afternoon in June last. Dr. McEwen informed me that the patient was about a week from term. The history of the patient was of considerable interest. Some months previously she had been seen by Dr. Donald, of Manchester, on account of excessive menstruation ; Dr. Donald then recognized several fibroid tumours, and, hoping to relieve the bleeding, he curetted the uterus. She was distinctly better as a result of this operation, and some little time afterwards became pregnant. The pregnancy progressed quite satisfactorily, except that the patient had a good many discomforts (sickness, vomiting, &c.), and required to be confined to bed for short intervals. About three weeks before I saw her she had an attack of hæmorrhage, not very severe however. This recurred at short intervals, and before I saw her there had been a pretty sharp bleeding. The fibroid tumours, which were situated entirely over the fundus, had become considerably larger. Suspecting placenta prævia, and in order to satisfy himself regarding that condition, Dr. McEwen examined the patient under an anæsthetic. He was then able to feel the placenta through the slightly dilated os, and, from the feel of the lower uterine segment, was inclined to the belief that it was centrally situated. This surmise of Dr. McEwen's proved to be correct. The case was then put before me by Dr. McEwen, and we discussed the treatment very carefully. Taking everything into consideration, I advised Cæsarean section and removal of the uterus. I came to this decision because I felt convinced that by ordinary methods of treatment for placenta prævia, I would almost certainly lose the child and subject

the patient to all the risks that we are all familiar with in connexion with placenta prævia. In addition, I felt that it would be hardly likely she would be able to retain her uterus much longer, and that possibly—nay, even probably—this was the last pregnancy she could have. Dr. McEwen entirely agreed with me, and I must here thank him for so cordially supporting me in the radical treatment I counselled. The whole nature of the case was explained to the husband, who, being an extremely sensible man, left the decision entirely in our hands. I therefore telephoned for my assistant (Dr. Dickie) to bring my abdominal instruments, and for Nurse Taylor, of the St. Elizabeth Home, to bring sterilized gauze, swabs, gloves, &c.

The operation was performed in the patient's bedroom and presented no great difficulty. I opened the uterus by the ordinary longitudinal incision and then clamped both broad ligaments. After removing the living child, I separated the placenta, which was implanted centrally over the os internum. I then removed the uterus by supravaginal amputation; the vessels were then ligated, the broad ligaments stitched over with catgut, and the cervix covered with peritoneum. The abdomen was then closed, and the patient put back to bed. One ovary was left; the other was cystic, and had to be removed.

The uterus contained a number of fibroids. The largest one on the anterior wall, about the size of a closed fist, bulged into the cavity of the uterus and underneath the peritoneum.

The patient made an uninterrupted recovery until the evening she got up for the first time, three weeks after the operation. She then developed a slight pleurisy, which continued for a few days, but soon subsided. Both she and the baby are in excellent health, and the other day she told Dr. McEwen that she had not felt so well for a great number of years.

I feel convinced that here was a case of placenta prævia in which Cæsarean section was the soundest treatment.

I have certainly seen one other in my private practice, where a similar treatment would have saved the child and in all probability the mother also. It was a case of central placenta prævia in a woman with a slightly generally-contracted pelvis, pregnant for the first time, and aged 36. With great difficulty she was delivered of a large child at full time. Version was performed to control the bleeding, and craniotomy was ultimately necessary before the child could be delivered.

Very judiciously chosen, I am convinced there is a place for Cæsarean section in certain cases of placenta prævia. By the ordinary methods of

treatment placenta prævia has a maternal mortality by 6·8 per cent., and a foetal of 50 per cent. to 60 per cent. in hospital practice; and the respective mortalities are much larger in general practice. In a carefully chosen case, at full time, not interfered with, and when there is every prospect of a difficult extraction if the child is delivered *per vias naturales*, would Cæsarean section have a higher maternal mortality than 6·8 per cent.? Certainly not; in fact, in series of cases where these respective treatments were employed, Cæsarean section could probably give a lower mortality, for I know of nothing more likely to be followed by death or grave septic mischief than a difficult extraction if the placenta is prævia.

But then there is the other individual concerned—the child. Undoubtedly Cæsarean section would give better results than the ordinary methods of treatment. Naturally one would not think of the operation unless the child was at or about full time.

I trust I am not misunderstood. All I claim in this short communication is that in certain well-selected cases at full time Cæsarean section has a place and is sound treatment both from a theoretical and practical standpoint.

DISCUSSION.

Sir WILLIAM SMYLY said that in the case reported, where placenta prævia was complicated by uterine myomata, Cæsarean section followed by hysterectomy was undoubtedly the correct line of treatment, and he most cordially congratulated Dr. Munro Kerr upon its success. Both conditions called for relief, and it was better practice and involved less risk to both mother and child to treat them simultaneously. He had not himself ever met with a case of placenta prævia in which he thought an abdominal operation advisable.

Dr. W. S. A. GRIFFITH had been convinced for some years that certain special cases of placenta prævia would be best treated, both to the interest of mother and child, by Cæsarean section. The difficulties, and therefore the dangers, were very slight where dilatation is easy and labour pains powerful, and the difficulties increased with the difficulty of dilatation and the absence of pains; and it is in the case where the placenta is so-called "central," the cervix closed and tough, with considerable hæmorrhage, that the danger is great. In January, 1905, he was called by Mr. Crabtree, of Weybridge, to a lady, aged 38, who had had two severe hæmorrhages, and found the child lying in the first position with the head above the brim. The cervix was closed and was thick and tough, the pelvis being of normal size. The vagina was narrow. Both husband and wife expressed the wish that the life of the child should be saved, if possible,

and readily agreed to the operation, which was at once performed. Both mother and child made an excellent recovery. The need of special care to ensure the complete detachment of the placenta impressed itself on Dr. Griffith. He had to remove a piece of firmly adherent placenta, which he had left behind, from the lowest part of the uterine cavity, as hæmorrhage occurred thirty-six hours after the operation. The boy is living and strong, and there has been no subsequent pregnancy. Vaginal section of the cervix, which in his opinion would be the operation of election in a similar severe case of accidental hæmorrhage, would probably involve too severe hæmorrhage in a case where the placenta was prævia.

Dr. HERBERT WILLIAMSON agreed that there were cases of placenta prævia in which the patient could be delivered more safely by Cæsarean section than by any other method. In such cases, when circumstances permitted, the operation should be performed. It was, however, seldom that such a procedure was called for. It was difficult to define the indications, but he would instance the case of an elderly primipara with rigid soft parts and no dilatation of the cervix. He had himself performed Cæsarean section for placenta prævia on one occasion. The patient was a primipara, aged 42, who in the seventh and eighth month of her pregnancy had several severe hæmorrhages, and three weeks before term was brought to town on an ambulance and placed under his care. She was very anæmic; the child was lying transversely in the left cephalo-iliac dorso-posterior position, and a fibroid the size of a man's fist was situated in the posterior wall of the uterus above the brim of the pelvis but below the child. He performed Cæsarean section and found a centrally-situated placenta prævia so firmly adherent to the fibroid that he was obliged to enucleate the latter in order to remove the placenta. He did not remove the uterus. The patient made an uninterrupted recovery from the operation, but, unfortunately, the child died twenty-four hours later.

The PRESIDENT (Dr. H. Macnaughton-Jones) said that the case brought before them raised the all-important question of the justification for Cæsarean section in placenta prævia. It had quite recently given rise to one of the most important discussions at the McDowell Centennial Anniversary in America, in which their late President (Dr. Herbert Spencer) took part. The case brought forward by Dr. Munro Kerr was obviously, from the myomatous complication, one in which the operation was indicated, as also in that mentioned by Dr. Williamson. Such complications demanded operation. He (the President) agreed that in a primipara with a rigid and undilatable cervix or abnormally small introitus, especially with centrally-attached placenta, the step was indicated. But he thought that many advocates of Cæsarean section greatly exaggerated the dangers of this operation to the mother. Henry Fry, in the discussion referred to, instanced the reports of 161 cases distributed between six operators, and also quoted Holmes, who had collected 1,029 cases distributed between eleven surgeons, and the mortality in both instances did not exceed 3·3 per cent. The whole question was one

well worthy of a special discussion when the vast experience gained in the British lying-in and maternity institutions could be availed of and an authoritative opinion might go forth from this Section. The well-known teachings of the Irish school, from which he himself hailed, and which for years he had followed, convinced him that only in very exceptional instances was Cæsarean section called for. As was stated by Fry, if we allow for the occurrence of placenta prævia once in 1,000 pregnancies, then the demand for Cæsarean section would not exceed one in 20,000. There were many incidental points which it would be well to have debated if the subject came up for special discussion in the Section.

Dr. MUNRO KERR, in reply, stated that he was much interested in what had been said by the previous speakers, and especially by the President. He had really brought forward this case because the subject of Cæsarean section in placenta prævia had aroused considerable interest in America and Germany, and he wished to obtain the opinion of the Society regarding this most important question. He was glad to hear that on the whole the Society approved of the operation in certain well-chosen cases, for he felt convinced that by Cæsarean section in well-chosen cases there would be less risk to the mother than if the ordinary methods were employed. He was quite sure, however, that the cases in which the operation was indicated were very few indeed.

**Points for the Use of, and Indications for, Electrotherapy
in some Gynæcological Affections.**

By J. CURTIS WEBB, M.B.

BOTH on the Continent and in America, electro-therapeutics and ionization have, for some considerable period, played a very important part in the treatment of certain gynæcological affections, and, as I can find no record of any paper on this subject having been read before this Society for a considerable time past, and as such great advances have, in recent years, been made in this department of therapeutics, I have ventured to think that my own experiences, which are now not inconsiderable in this special treatment, might not be without interest. It will be well briefly to consider (a) The currents used and their action on living tissue; (b) Special instruments necessary for their employment, and the methods of using them; (c) Some of the commoner gynæcological diseases suitable for treatment by electrotherapy and the methods of treatment in each class of case.

(a) For the purposes of gynæcological treatment currents can be divided into the induced, the constant, and the static. The first, if the interruptions be very rapid, and taken from a long, fine-wire secondary coil, have a sedative effect. Exactly how this sedation is brought about we do not know, and therefore the treatment is somewhat empirical; but I have satisfied myself as to its effect in many neuralgic affections of the pelvic organs. Where it is desired to produce contraction of muscular tissue in cases where the symptoms depend on relaxation, we must use an induced current of slow interruptions, allowing time for the muscle fibre to relax between each such contraction; otherwise a state of tetanic spasm is produced, and the muscle is only tired out without being strengthened. The interrupted or induced currents are of but secondary importance when compared to the direct current, which is really our sheet anchor in gynæcological electro-therapeutics. The action of this current is threefold: the chemical polar action, the ionic action, and the interpolar action. The chemical polar action need only be considered when we are dealing with bare metallic electrodes, and must be discussed in conjunction with the ionic action. When the metallic electrode—e.g., such as is introduced into the uterus—is connected to the positive

pole of our source of current, the metallic particles of which this electrode is composed are driven to a certain depth into the adjacent living structure in the form of ions, and these ions, in the case of a zinc electrode amalgamated with mercury, have a strong antiseptic action. At the same time dissociation of the fluids of the organ in contact with the electrode takes place; the acid radicles are attracted to the region of the electrode and produce a cauterizing and hæmostatic effect; while the basic radicles, like the metallic ions above considered, are propelled further into the tissues. Thus the cauterizing effects in the region of a bare metallic positive pole are really due to ionization of the component elements of the body fluid—i.e., lymph and blood-plasma, or, for practical purposes, solution of sodium chloride. On the other hand, if the metallic electrode be negative, there is no diffusion of metallic ions and the basic alkaline components of the body fluid are drawn to the neighbourhood, producing a solvent or congestive effect on the tissues. For the above reasons it is found that, after treatment, a positive electrode is often difficult to withdraw, being somewhat adherent to the eschar, while a negative electrode has to be carefully held in place during the application to prevent its slipping out. In the case of vaginal electrodes, which I usually employ in the form of cotton-wool pledgets surrounding the ends of metallic rods, the rest of the shafts of which are insulated, no ionization of the metallic particles takes place, and there should be no chemical local action; but there is ionic diffusion of the constituents of the salt, in the solution of which the pad has been soaked. By this means iodine ionization may be employed in cases of pelvic effusion, &c.

This very short account of ionic and chemic polar action is all that can be touched on in a paper of this length. In regard to interpolar action, a factor on which I would place great emphasis, its effects can be traced “to the influence upon nutrition of the chemical interchanges that occur throughout the circuit in the onward progress of the electrons that appear finally at the poles; to the influence upon nutrition of the circulatory changes that result from vasomotor stimulation; and to the contractions produced in unstriated muscular tissue by the passage of heavy currents, even at a distance.” (Massey.)

So much, then, briefly for the currents used and their methods of action. It will be noticed that I have made no mention of sinusoidal currents, as I have not found their effects in any way superior to induced currents, and the apparatus to produce them is much more cumbersome. Neither do I propose here to enter on the subject of treatment of static electricity, though I have found it of immense value in the cases of

dysmenorrhœa in virgins, and almost a specific in those cases of lumbar pain so often associated with some derangement of the female pelvic organs. I expect, however, that few here have static machines or the time to use them, and I am anxious to bring before you methods of treatment that *all* can use.

(b) The special instruments necessary for the applications of electricity in gynæcological practice are not many. An efficient source of current is required, either a dry or wet cell battery of twenty to thirty cells, or a switchboard with which to use the current from the mains, either being furnished with a milliamperemeter, reading from 1 ma. to 250 ma., and a rheostat that will allow of the current being turned on gradually from zero to the required strength. For the faradic current I prefer the coil made by Messrs. Gaiffe, as it gives rapid or slow interruptions at will, and one can use a short coarse or a long fine wire secondary, according to requirements. For the indifferent electrode one can use large pads of gamgee well soaked in warm water and soaped, or moist clay pads. I prefer the latter, as there is no risk of wetting a patient's clothes. In either case they must be closely applied to the skin, and a plate of copper attached to the required pole placed on the outside. For uterine electrodes, I employ a kind made by Messrs. Gaiffe, or a type made for me by the Medical Supply Association. These I consider better, as the current is more concentrated at the bare end, and they are easily sterilized by being kept in one in twenty carbolic for an hour or two before use. I always use zinc in preference to copper, which some users advocate, saying the ions of copper are more hæmostatic than those of zinc. This, however, I have failed to corroborate, and when an ionic effect is desired—i.e., when the internal electrode is positive—I always amalgamate the zinc portions by dipping them into 10 per cent. H_2SO_4 , and then into metallic mercury. For vaginal electrodes I use a zinc rod, the end of which is surrounded with a pledget of wool covered by chamois leather tied in place by thin silk ribbon, and the ends left long. The rest of the rod is covered with rubber tubing. It is well also to have an Apostoli bipolar electrode for faradic applications to the vagina.

Having thus very briefly sketched the mode of action of the ordinary currents employed in gynæcological ailments, and the instruments satisfactory for their application, there is left the most important part of the paper—viz., the type of cases suitable to electrotherapy—and I think it will be more interesting and convincing if I confine myself to a consideration only of those diseases in which I have myself had bene-

ficial results. The curative effects of electricity are claimed for many ailments in which I have not had an opportunity of trying it, and therefore of its effects in such ailments I am not in a position to speak. I have not seen large fibroid tumours emulate the snark and "softly and silently vanish away," neither have I seen ectopic gestations absorb and disappear under its influence, as has been reported by some writers. I have not tried zinc-mercury cataphoresis in cases of malignant disease, though I am willing to admit the possible—nay, even probable—benefits that might arise from so powerful a means of destruction of tissue and sterilization of neighbouring parts in that class of case before which we stand at present comparatively powerless, and which we term inoperable.

The types of cases in which satisfactory results from electrical treatment can most readily be obtained are those in which the disease seems likely to be benefited by one or more of those actions which, as I have shown, electric currents have on living tissue—that is to say:—

(I) With faradic currents: Either sedation or muscular stimulation and contraction, according to the strength and rapidity of interruptions of the induced current.

(II) With the constant current: (*a*) The cauterizing, hæmostatic action that occurs at the positive pole; (*b*) The congestive or liquefying action at the negative pole; (*c*) The diffusion of ions of the constituent of which the electrode, positive or negative, according to circumstances, is composed; (*d*) The effect on metabolism that is brought about by the interpolar action—that is to say, the results on the tissue of the flow of the current *per se*.

(III) With the static current: (*a*) The cellular vibration that occurs during an application of the wave current, causing a dispersion into the neighbouring lymphatics of the products of inflammation; (*b*) The increased supply of blood to the parts caused by the brush discharge; (*c*) The constitutional effects, viz.: (1) lessening pulse-rate; (2) increase of pulse volume; (3) lowering of tension; (4) deepening of respiration and increased elimination of; (5) lessening of nervous irritability and production of drowsiness; (6) a general diaphoresis and an augmentation of the quantity of solids in the urine.

DISEASES OF THE VAGINA AND VULVA.

In inflammatory diseases of the vagina and vulva, characterized by pain, tenderness, and leucorrhœal discharge, whether they be due to bacillary infection or not, the action of copper or zinc ionization will

very greatly assist any other means that may be employed in bringing about a cure. Naturally such means as rest, douches, and vaginal medication are not to be neglected, but the stimulating action of the current *per se* and the antiseptic penetrating effect of suitable ions will very soon cure this class of case, which is notorious for its obstinacy. Perhaps the best means of using this form of treatment is by means of Dr. Sloan's vulvo-vaginal electrode.¹ For those who do not possess this appliance I would advise an electrode such as I show here. It consists of a vulcanite tube with perforations at the sides, passing down which is a metallic wire, or spring, with a suitable arrangement for attaching the connecting cord from the source of current, and also the means of affixing the indiarubber pipe of a douche-can. Over this vulcanite tube is slipped a piece of sausage-skin, rendered sterile by boiling or soaking in antiseptic lotion, and which has been closed at one end and invaginated, the other end being tied round the vulcanite tube. This bag of membrane can now be inserted into the vagina and filled from the douche-can with the solution of the salt it is desired to ionize, so as to distend the whole passage. If this piece of apparatus is now connected to the proper pole of a battery or other source of current, the other pole being connected to a large electrode placed on the abdomen or back, and a current passed up to the limit of tolerance for ten to thirteen minutes three times a week, the required ions will, owing to the sausage-skin being permeable to the fluid, be driven into the vaginal mucous membrane.

DISORDERS OF MENSTRUATION.

(1) *Amenorrhœa*.—When this occurs in virgins it can often be relieved by abdomino-dorsal applications of the constant current, using up to 100 ma., a large pad being placed over the lower dorsal region (positive) and a similar pad (negative) over the hypogastrium. At the same time, of course, attention must be directed to improving the patient's general health by appropriate remedial measures.

(2) *Scanty Menstruation*.—In the cases of this trouble arising in women who have borne children, excellent results can be obtained by the employment of a negative intra-uterine current, using about 10 ma. to 30 ma. for ten minutes once a week, during an intermenstrual period, with bipolar faradic every other day. I have in my mind the case of

¹ *Proc. Roy. Soc. Med.*, 1909, ii (Electro-Therap. Sect.), p. 123. and *Lancet*, 1909, ii, p. 68.

a lady, aged 43, who for twelve years had suffered from scanty menstruation, the flow only lasting one day as a rule. This condition came on after an attack of peritonitis, and I found a fixed retroflexed uterus. She consulted me in 1905 for the terrible headaches and sensations of congestion and throbbing of the veins of the head and neck that came on at each period and lasted about four days, leaving her a "wreck" for a week after. A course of three weeks' treatment, as above described, kept her free from headaches for over six months, and resulted in each period lasting four to six days. I now give her two treatments, just before a period, three times a year, and she keeps well and comfortable and very rarely gets headaches or feelings of congestion.

(3) *Dysmenorrhœa*, which I hold to be due to a tropho-neurosis, giving rise to a muscular spasm, or cramp, at the time of the period, requires for its successful treatment some means of co-ordinating these irregular uterine contractions, and, if we accept Dr. Herman's theory of imperfect development of the spinal and sympathetic centre, of stimulating this centre to full function. Many women suffer great inconvenience, and often days of torture, from this complaint, but I have had most satisfactory results from the following plan of treatment: I first apply the wave current from the negative side of a static machine, by a long metal electrode passed up the rectum and pressed against the posterior wall of the uterus for about fifteen minutes. The procedure is absolutely painless—hardly even uncomfortable. The constant current is then employed, and here the method of application varies, according as one is dealing with a virgin or a married woman. In the former case the method of procedure is usually as was described under amenorrhœa, with the object of stimulating the spinal sympathetic centres and of improving the metabolism of the uterus itself. Three such treatments a week for one or more intermenstrual periods will often entirely relieve the patient from pain for many months, and should there be any signs of its return, one or two treatments, just before a period, will be sufficient. When the sufferer is a married woman the static wave current is used three times a week, as above; but one is able to apply the constant current immediately to the uterus by connecting both the abdominal and dorsal pads to one pole—usually the positive—and inserting a metal electrode connected to the negative pole inside the organ. In such a case we get the interpolar action of the current as well as the congestive effect of the negative pole to the surface of the uterine cavity. About 20 ma. every fifth or sixth day for one or two months will usually bring about a most satisfactory result. In those

cases of dysmenorrhœa in which the ovaries are involved, and where there is congestion and tenderness of the ovaries on vaginal examination, the treatment must consist solely of abdomino-vaginal applications till all signs of ovarian irritation have disappeared. Then one may cautiously commence with intra-uterine treatment, as above described.

DISEASES OF THE UTERUS.

In any inflammatory diseases of the above organ, applications of the constant current and the effects of zinc-mercury ions will give the most brilliant results; and, further, in those cases of uterine hæmorrhage, either in the form of menorrhagia or metrorrhagia, due to endometritis, metritis, fibroids or a fibrotic condition of the organ, subinvolution, or ulcers on a uterus in a state of complete procidentia, I know of no treatment to equal that now under discussion. In my experience, in any of the above conditions it takes the place of curettement, over which most popular operation its advantages are manifold; for while the curettement only removes the mucous membrane and does not affect in any way the underlying muscular layers, in zinc-mercury ionization from the positive pole we obtain: (a) destruction of the diseased mucous membrane and arrest of hæmorrhage by the cauterizing action; (b) penetration into the subjacent layers of tissue of the antiseptic ions; and (c) the general improvement of this subjacent muscular layer by the interpolar electric action. Further, ionic treatment is painless, does not require an anæsthetic, and does not confine the patient to bed. The technique of treatment is similar to that already described for the intra-uterine applications, and it will be easy from previous considerations of their respective effects to decide on which pole should be the active one. Let me note two or three cases that I have myself treated:—

(1) Mrs. K., aged 45, was seen in April, 1906. She gave a history of a fibroid for the past nine years, menorrhagia for eighteen months, and constant pain on right side on exertion. The uterus measured three-quarters of an inch over the normal, and there was a fibroid as big as a tennis-ball on the anterior and right side. Between April 18 and June 2 she had seventeen treatments of ZnHg ionization. About a year ago she told me that since the treatments there had been no excessive catamenial loss and no pain on exertion, unless she got very unduly tired. The size of the fibroid had not altered.

(2) Mrs. S., aged 49, was sent to me in January, 1908, by Dr. John Phillips for very profuse menorrhagia and metrorrhagia for the past three years, and at that time the hæmorrhage was practically continuous. I gave her fifteen

treatments between January 21 and February 19, and the bleeding had ceased after the third, and did not return. Up to June she had normal periods, lasting four or five days and not excessive, but in June one lasted eight days: so she had six more treatments. Since then there has been no return of hæmorrhage; she is able to bicycle and lead a normal life. The menopause occurred last February, a year after the first course of treatment ceased. The sound passed 1 in. beyond normal, and the diagnosis was one of general fibrotic condition of uterus.

(3) Mrs. C. B., aged 32. Backache, leucorrhœa, and catamenia lasting six days, with clots since birth of child in India, eighteen months ago. *Per vaginam*, os very patulous, sound passed $1\frac{1}{2}$ in. beyond normal, uterus retroverted, and a clear discharge coming from the os. Diagnosis—subinvolution with endometritis. ZnHg ionization and static wave to back effected a complete cure.

(4) Mrs. B., aged 45, had profuse periods ever since puberty. Has had four children and several miscarriages, the last four years ago. Of late, periods have been very profuse, lasting seven to ten days, and hæmorrhage coming in gushes. After the periods were over she used to get palpitations and dyspnœa. The patient had fifteen treatments with zinc ionization, beginning last February. In July she reported that she had not been kept in bed by any period since the treatment, that she had been to several balls when the catamenia were on with no ill effects, and that the loss had not been excessive.

(5) A patient with subinvolution after the birth of a child in 1905 came to me in June, 1908, complaining of ovarian pain with menorrhagia and menorrhagia. There was uterine discharge and the sound passed 1 in. beyond normal. Under ionization and monopolar faradic applications all the symptoms disappeared, and the patient has remained quite comfortable up till now.

In diseases of the tubes and ovaries, if of simple inflammatory nature, the treatment under consideration can do much. The method of application varies with every case, but, speaking generally, abdomino-vaginal applications should precede, for some time, any intra-uterine treatment, especially if there be much local tenderness. A method that I have found beneficial in several cases of ovaritis, non-purulent salpingitis, and also in one or two cases of pelvic inflammation, is to paint the fornix on the affected side with equal parts of lin. and tinct. iodi, and then to apply the vaginal electrode soaked in 2 per cent. solution of K.I. and attached to the negative pole every other day, passing about 20 ma. for twenty minutes. When there are signs of the resolution of the inflammation of the appendages, then attention should be directed towards the endometritis, which, as we know, is practically an invariable accompaniment.

An instructive case was that of a young woman, an artist, who was prevented from following her profession because of the great pain over the left ovarian region brought on by standing. On vaginal examination the left ovary was found enlarged and very tender. The application of abdomino-vaginal and bipolar faradic currents effected a cure over two years ago which is permanent as yet. I may add that she had tried systematic hot douching and vaginal medication, and she had been told that she ought to submit to ovariectomy.

One more case may be of interest. A lady, aged 44, staying in London, came to me in September, 1908, with a history that in May, 1907, she had a swelling on the left side of the uterus, with acute pain and a temperature of over 100° F. for three weeks. Two months after the beginning of the illness there was a sudden white discharge for twenty-four hours. This occurred again in December of the same year, lasting five days, and again in March, 1908, and ever since the beginning of her illness she experienced acute pain in the lower part of the left side of the abdomen, and backaches if she walked, or got tired, or stooped. *Per vaginam*, the left tube felt much thickened, but not tender, and there was some leucorrhœa. In October I began treatment with abdominal vaginal constant current applications and vaginal faradic and static wave to back. By the end of October she was feeling much better, and I added intra-uterine treatment, and she left London in the middle of November. A few days ago I heard that she had had no pain since the treatment and could now walk about all day and play a little tennis.

This paper has already extended to greater lengths than I intended, and yet I am fully aware that I have but touched the fringe of my subject. As far as I have gone, I have convinced myself of the enormous value of this form of treatment in many of the pelvic diseases of women. Patients can by this means, in many cases, be cured without operations and without being doomed to bed and a more or less prolonged convalescence, when, without it, nothing but operation remains for them. My object in reading this paper is to try to induce members of our profession, both general practitioners and gynæcological specialists, to take a greater interest than would seem to be the case up to now in a most powerful means of relieving and curing many of the ailments from which women suffer; and may I implore those who are attached to a hospital to investigate this mode of treatment to its utmost extent, that the reports of their fuller researches may save many a woman from a mutilating operation or a more or less ineffectual curettement.

DISCUSSION.

Dr. LEWIS JONES considered that, apart from the general interest of the paper, there was a special interest for gynæcologists in that part of the paper which dealt with ionic medication. The introduction of drugs by the aid of electricity might, he considered, find a place in gynæcological work, and the method had been advocated by Dr. Margaret Cleaves, of New York, and by Dr. Samuel Sloan, who had communicated a valuable paper to the Electro-Therapeutic Section a few months ago.¹ In particular he would say that where the indications were for the introduction of an antiseptic into the mucous membrane, at least to a depth of 2 mm. or 3 mm., the use of zinc or copper ions might confidently be tried. He regretted that his experience of ionic medication in gynæcological cases was not sufficient to permit of his quoting specific cases occurring in his own work, but from the general principles underlying ionic application he felt sure that the methods advocated by Dr. Curtis Webb were worthy of trial.

Mr. SPANTON expressed his surprise that in discussing the different forms of electric treatment in gynæcology no mention had been made of the name of Dr. Apostoli, who certainly deserved credit for being one of its chief pioneers. Mr. Spanton had adopted his treatment in cases of uterine myoma with hæmorrhage with most excellent results twenty years ago, but found the method somewhat cumbersome and difficult to carry out in hospital practice, and expensive and tedious in private patients, and these were probably the chief reasons why it had somewhat fallen into disuse.

Dr. AMAND ROUTH said the Section was indebted to Dr. Curtis Webb for bringing before them some electro-therapeutical methods of dealing with gynæcological troubles in women who were averse to "operations," or were for some other reason unable to be dealt with in the usual way. Some years ago, when he had more time, Dr. Routh adopted the methods advocated by Apostoli, and he had found real benefit from the use of the positive intra-uterine pole with a current of 60 ma. to 120 ma. in cases of uterine hæmorrhage due to endometritis, or fibroids, or subinvolution. He felt, however, that the time, trouble, and expense of the treatment was not justified by the results obtained as compared with those following ordinary methods of treatment. In severe cases of chronic subinvolution and endometritis curettement was preferable, and accomplished much more than mere removal of hypertrophic mucosa, as suggested by Dr. Webb; for it led to radical changes in the fibromuscular tissues of the uterus, promoting absorption of inflammatory exudations, lessening bulk, and, generally speaking, restoring the uterus to its normal size and function. Fibroids were only symptomatically cured, and the cure was only permanent when near the menopause. He felt more inclined to believe that the treatment of fibroids by X-rays, as advocated by Albers-Schönberg, would cause their atrophy and cure much more effectually than the electrical treatment, even if associated

¹ *Proc. Roy. Soc. Med.*, 1909, ii (Electro-Therap. Sect.), p. 123.

with ionization, as carried out by Dr. Webb. The fact that Graafian follicles had been proved to disappear from rabbits' ovaries in twelve days, and that sterility had been proved to follow the use of X-rays, and that rapid shrinkage of fibroids and cessation of hæmorrhage in women had been observed by Crabbe and Caldwell of New York, led us to hope that we were on the eve of finding some reliable method of treatment as an alternative to hysterectomy. He believed also in the germicidal action of high-frequency currents in septic pruritus pudendi, and would be glad of an opportunity of trying it by means of a glass intra-uterine electrode in a case of septic puerperal endometritis. He hoped that in time the cases which gynecologists could safely and hopefully hand over for treatment to their colleagues in the X-ray and electrical departments of medicine would become defined, for there was abundant room for any remedies which could take the place of radical operations.

Dr. HERMAN STEDMAN said that he had selected some half-dozen cases of chronic endometritis and cervical catarrh and had endeavoured to cure the discharge by means of ionization with zinc and copper, but that after six treatments of 600 ma. minutes, had only in one instance succeeded in effecting a cure. The dose used was the standard adopted on the Continent, and was arrived at by using the strongest current the patient could bear, and maintaining it for a sufficient time, which, when multiplied in minutes by the number of milliamperes, resulted in a total of 600 ma.—e.g., 30 ma. for twenty minutes

600 ma. m. [The author replied that he used a current of 100 ma. to 120 ma. for ten to fifteen minutes.]

Dr. F. HOWARD HUMPHRIS wished to bear out the remarks of Dr. Curtis Webb, and from personal experience to testify that, as an adjunct to gynecological treatment, electrotherapy was invaluable. He had used it himself for several years, and had found that results could be obtained from its use which he had been unable to get without it. Various ill-defined pelvic pains and backache could be relieved as surely with the faradic current as with a hypodermic injection of morphia, only it was not with every faradic coil that these results could be obtained. Different lengths of wire were needed in different cases, but in most conditions in which a sedative effect was required, a long fine-wire coil, used with a bipolar vaginal electrode, would give relief in fifteen to twenty minutes. In cases of subinvolution, in prolapse, and in all conditions in which the uterus was lacking in tone, electricity was one of the most valuable remedial measures. In these cases, static electricity, the vacuum tubes applied locally and the wave-current over the lumbar region, Dr. Humphris had found to be of special benefit, pain being relieved from the first, and the pathological cause being ultimately improved or replaced by the former physiological healthy condition. Dr. Humphris heartily concurred in the statement as to the value of the incandescent-light bath, but preferred the use of the single high-candle-power lamp, especially in cases of painful menstruation, and wished that time had permitted him to quote several successful cases. In cervical endometritis Dr. Humphris had been in the habit of using the uncovered copper electrode in

the cervix, with the indifferent negative pole made of clay, thus by cataphoresis disinfecting the cervical canal with nascent oxychloride of copper. This, he explained, is the procedure of Dr. Betton Massey, under whom he had worked for a little time, and to whose teaching and book¹ he owed a deep debt. In conclusion, Dr. Humphris supported the plea in the valuable paper of Dr. Curtis Webb for the more general use of electricity as a therapeutic agent in gynæcological practice, not as a universal panacea, but as a most useful addition to the usual means of resource; not necessarily as a substitute for, but as an adjunct to, the more classic forms of treatment; as a palliative in many cases of pain, and as a curative agent in those cases which are amenable to its influence.

The PRESIDENT (Dr. Macnaughton-Jones) said that years since Charles Goodell had taught him that there were things in gynæcology we had to "learn to unlearn"—practices we followed that we had to abandon; others we despised which we were forced to accept. The one thing we all had to unlearn was bias and prejudice. A good deal of both had existed with regard to electrical treatment in gynæcology. This might be explained by the fact that we had hitherto been generally ignorant of the nature and structure of electricity and how the different kinds operated in their therapeutical applications. Physicians desiring to be exact looked with suspicion on a treatment which they regarded as empirical, and the action of which they could not explain. Hence a bias and scepticism prevailed, but this should not prevent the acceptance of new facts and the adoption of a more rational method. The late Dr. Charles Routh, almost simultaneously with Cutter, in the sixties, advocated electrical treatment; then came Tripier, and following him Apostoli. When the latter's methods and results were published, he (the President) had procured all his appliances and had tried both electrocausis and the faradic treatment. For the latter he used different coils and the high-tension battery, with the various sized coils recommended by Apostoli, and which had just been referred to, with the vaginal and intra-uterine bipolar electrodes. With regard to electrocausis, he had so serious a result in one case that he abandoned it altogether. The patient was a lady who suffered from cataleptic and hystero-epileptic attacks, associated with profuse hæmorrhages from a fibromatous uterus. Every practical precaution was taken in the application of the current, both as to strength and antisepsis. At first she apparently did not suffer from the treatment. A skilled electrician was present at each sitting, which was given in bed; suddenly, she developed symptoms of peritonitis, which proved fatal. Apostoli pointed out that this class of patient was peculiarly susceptible, and the most riskful to treat. With regard to faradization in amenorrhœa and dysmenorrhœa, he had varying results both with the constant and faradic currents. In many cases, as might be expected, there was no influence on either the flow or the pain. The present discussion had, however, rather turned on cataphoresis and ionic medication. Of this he (the President) had

¹ "Conservative Gynæcology and Electro-Therapeutics." G. Betton Massey, M.D. F. A. Davis Co., Philadelphia.

no personal experience in gynæcological cases. There was no doubt in his mind, from the reports he had read of the results in other hands, that this method of treatment might be very beneficial in certain affections of the female genitalia, but he quite agreed with Dr. Routh that there were cases in which the delay necessitated in carrying out the treatment might be disastrous to the patient. He could not agree that curetting only removed the mucous membrane. It affected intimately the underlying muscular structure, and set up a new circulatory relation between the uterus and adnexa through the blood-vessels and the lymphatics, having a most important effect on the latter. Of course, he referred to thorough curetting, and at times tamponading of the uterus, not to mere superficial and so-called scraping. The all-important point, in deciding on any form of electrical treatment, was diagnosis. Apostoli had emphatically insisted on this. In many forms of adnexal disease such treatment was not only utterly inapplicable and useless—as, for example in pyosalpinx—but extremely dangerous, not merely from the point of view of delay, but from the nature of the interference itself.

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Obstetrical and Gynæcological Section.

November 11, 1909.

Dr. H. MACNAUGHTON-JONES, President of the Section, in the Chair.

Leucoplakic Vulvitis and its Relation to Kraurosis Vulvæ and Carcinoma Vulvæ.

By COMYNS BERKELEY, M.B., and VICTOR BONNEY, M.S.

THE occurrence in the tongue of a peculiar type of inflammation leading on to a leucoplakic condition of the mucous membrane, and terminating in carcinoma, has long been known. It was first described in 1869 in the *Transactions of the Clinical Society* by John Whittaker Hulke, the Council of the Royal Medical and Chirurgical Society having refused to publish his paper some four years previously. Sir Henry Morris described the second case, and also was the first to call attention to the frequency with which a similar type of inflammation preceded carcinoma of the vulva, and operated upon several patients with the combined disease. Both of these eminent surgeons were members of the Middlesex Hospital surgical staff—a matter of great interest to the authors of this paper. It is remarkable that a clinical observation of such importance should have attracted so little attention, especially by gynæcologists, and that the only two English writers who have subsequently drawn attention to the subject—namely, Bland-Sutton and Butlin—should both be general surgeons.

The relation borne by leucoplakic vulvitis to carcinoma is closer than that of any other pathological lesion, with the exception of the entirely modern X-ray dermatitis. In 1885 Breisky drew the attention of gynæcologists to a disease which he named kraurosis vulvæ. His description led to much confusion, for whilst he undoubtedly described a till then unrecognized condition, subsequent writers on the subject down to the

present time, as can be seen from the text-books, appear to have confused the conditions of leucoplakic vulvitis and kraurosis vulvæ, and to have considered the different phases of one and the same disease.

In this paper, then, we shall first proceed to show that leucoplakic vulvitis and kraurosis vulvæ are two distinct diseases, and we shall then pass on to consider the relation borne by the former to carcinoma of the vulva.

LEUCOPLAKIC VULVITIS.

Definitions.—Leucoplakic vulvitis is to be defined as a chronic inflammatory condition of unknown origin characterized in its early stages by marked hyperæmia and cellular activity, and in its later phases by marked epithelial hypertrophy and a thickened sclerosed and retracted condition of the subepithelial tissue.

Distribution.—Its distribution varies with its severity and time of incidence. In a well-marked case the whole of the vulva may be implicated, with the exception of the vestibule and orifice of the urethra, which are never affected; and not only may the labia majora and minora, anterior and posterior commissures and the clitoris suffer, but the disease may even spread laterally to the folds of the thighs and posteriorly over the perineum and skin surrounding the anus.

Signs.—Clinically, leucoplakic vulvitis may be divided into four stages, according to the macroscopical features which it presents. In the first stage the parts affected have a reddened, swollen, and excoriated appearance, and their surface is dry. In the second stage, whilst the labia minora decrease in size and may even become mere ridges, there is a subepithelial thickening so that the parts can be said to have undergone "retraction with thickening." The colour of the affected area now changes from red to white of a semi-opaque character. This opacity becomes intensified in patches at first, and later on over the whole of the diseased surface, a white appearance resulting from which the disease obtained its name of leucoplakia of the vulva. In the third stage, where the disease is most marked, cracks and ulcers may make their appearance, the ulcers giving rise to a slight discharge and bleed sometimes when touched. At this stage carcinoma quite commonly becomes engrafted on some portion of the diseased area, and, as a rule, it commences in the ulcer or fissure. If the patient escapes the incidence of carcinoma, a fourth and final stage supervenes in which the vulval surface is smooth, shiny, and white, and the labia minora and

clitoris practically disappear owing to the contraction of the subepithelial tissues, and the disease becomes quiescent.

Symptoms.—The striking and often the only symptom of leucoplakic vulvitis is pruritus, which is peculiarly intense in the first and second stages. In the third stage the pruritus is more or less masked by the pain and acute sensitiveness due to the exposure of the nerve endings in the floor of the ulcer or crack. In the fourth stage the symptoms disappear, and the patient ceases to complain. If carcinoma supervenes at the third stage the ulcers become very painful and tender, bleed easily on being touched, become hard and indurated in spite of local treatment, and enlarge fairly rapidly.

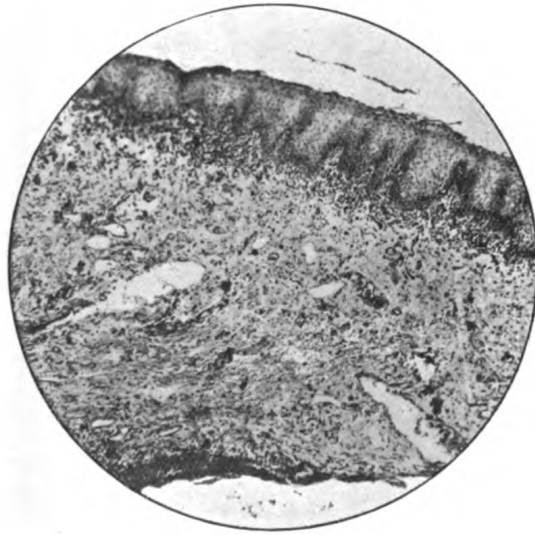


FIG. 1.

Earliest stage of leucoplakic vulvitis. Surface desquamation of epithelium and diffuse subepithelial lymphocytosis.

Pathology.—The pathology of leucoplakic vulvitis may be conveniently divided into stages. Such a division, however, bears no reference to duration, but is governed solely by the histological features which the diseased tissue presents. It is necessary to emphasize this, because different parts of the diseased area may be respectively in different pathological stages at the same time. From a study of a great number of sections obtained from twenty-four cases operated upon by us, it has been possible to construct an account of the full gamut of histological changes which characterize the disease from its initiation to its subsidence. In the

first stage the epithelium is swollen (fig. 1), and the individual cells less firmly attached to one another, so that the desquamation of the superficial cells is excessive. The subepithelial tissue is abnormally vascular and is crowded with lymphocytes, some of which have intercalated themselves between the cells of the basal layers of the epithelium. Few or no polynuclear leucocytes are to be seen, and the fixed tissue cells of the part have not proliferated. In the next stage the type of the subepithelial tissue-cell proliferation begins to alter. Lymphocytes are still conspicuous objects, but besides these are groups of plasma cells and an increased number of large hyaline fixed connective-tissue cells normal to the part. The sustentacular elements become altered, the white fibres

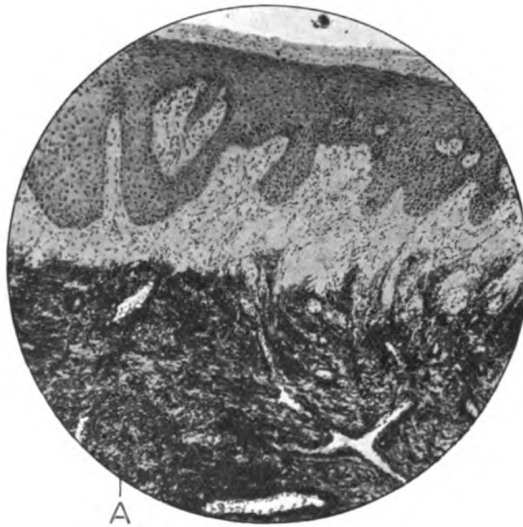


FIG. 2.

Second stage of leucoplakic vulvitis. A hyaline zone of de-elasticized tissue underlies the epidermis; beyond this area are shown the elastic fibres (stained darkly) which are normally found in the connective tissue. The interpapillar epithelial processes are elongated. A new lymph node is seen embedded amongst the elastic fibres at **A**.

become decollagenized and more hyaline in appearance; whilst the elastic fibres have in many places completely disappeared over a certain subsurface area, so that a definite zone of de-elasticized and hyaline-looking tissue intervenes between the epithelium and the normal tissues deeper down (fig. 2). That many or all of the lymphocytes seen in the microscopic field are produced in the diseased area is proved (1) by the absence of these cells in the cross-sections of blood-vessels, and (2) by

the appearance at this stage of definite new lymph nodes in the sub-epithelial tissue which contain germinal areas showing karyokinesis, and are not normally present there (fig. 2). The appearance of these new lymph nodes is very interesting, because they are commonly found in the neighbourhood of carcinoma, to which disease the condition under discussion bears, as already stated, a very close relation. The origin of the plasma cells is undoubtedly local. They appear to be developed from certain elongated nuclei found in resting connective tissue. The large hyaline fixed tissue cells proliferate both mitotically and amitotically from similar cells normally found in the part, and the same may be said of endothelial cells.¹ The epithelium at this stage is markedly

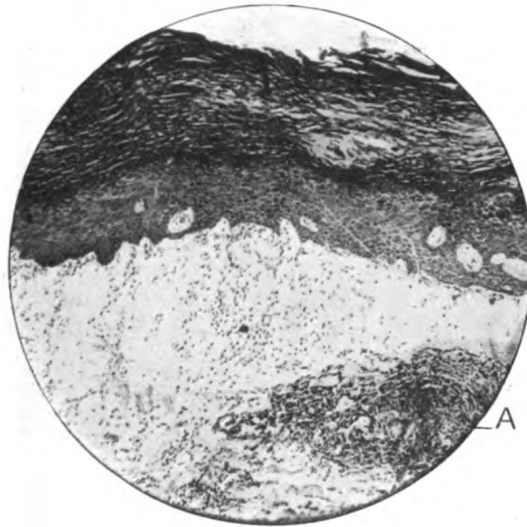


FIG. 3.

Third stage of leucoplakic vulvitis. The superficial epithelial cells have undergone extensive hypertrophy and keratinization. The interpapillar epithelial processes are atrophying owing to the subepithelial fibrosis which has taken place. A new lymph node is seen at A.

hypertrophied, and many karyokinetic figures can be seen in it. The hypertrophy is chiefly of the basal layers, and the interpapillar processes of the epithelium are much elongated. The surface cells, on the other hand, still show a tendency to early desquamation, which reaches its

¹ One of us has lately discussed fully the origin of these various groups of inflammatory cells—Victor Bonney, "Hunterian Lectures," Royal College of Surgeons; Seventh Report of Cancer Research Laboratories of the Middlesex Hospital, 1908; and *Lancet*, 1908, i, pp. 1389, 1465, 1535.

maximum in the numerous shallow ulcers and fissures that beset the diseased area. At these points so great has been the shedding of the superficial epithelium that the apices of the elongated papillæ of the corium almost reach the surface. It is also noteworthy that the inflammatory cells already described—and particularly the lymphocytes—tend to aggregate around the tips of the interpapillar epithelial processes. In the third stage, the cellularity of the subepithelial tissue begins to give place to fibrosis (fig. 3). Many of the inflammatory cells disappear; collections of lymphocytes and plasma cells are still scattered over the field. A re-deposit of collagen occurs around the fibroglia fibres of the fixed tissue cells, which, in the preceding stage, have been greatly in-



FIG. 4.

Final stage of leukoplakic vulvitis. The epithelium is thinner than normal; early keratinization of the surface cells is occurring. The subepithelial tissue is diffusely sclerotic.

creased in number, so that the tissue, from being unduly rarefied, now becomes denser than normal. The hypertrophy of the epithelium is still maintained, but the growth tendency now appears to be directed upwards, resulting in the formation of a dense mass of keratinized squames, whilst the interpapillar downgrowths are disappearing, so that the epithelium rests on the sclerosed subepithelial tissue more as a flat sheet. There is a total absence of yellow elastic tissue in the sclerosed subsurface zone. In the final stage (fig. 4) the sclerosis of the

subepithelial tissue is complete. Very few cells are to be seen under the epidermis. Such as are present are of the fixed connective-tissue type. The subepithelial zone of de-elasticized tissue is of less depth, but denser. The epithelium, probably starved by the increasing fibrosis going on underneath it, becomes thinner, whilst the cells show an early tendency to keratinization. Clinically, at this stage, the disease is quiescent. The surface of the vulva is smooth and shiny white in appearance, and the elevations of the labia minora and clitoris have become flattened down almost to disappearance, owing to the contraction of the subepithelial tissue. Pruritus has ceased, and the patient ceases to complain.

KRAUROSIS VULVÆ.

We have now to contrast leucoplakic vulvitis with kraurosis vulvæ. Since Breisky's original paper the most perfect clinical description of this latter disease is that of Thibierge, with whose conclusion we are in complete agreement. Kraurosis vulvæ consists of an atrophic condition of the vulva associated clinically with stenosis of the vaginal orifice and pathologically with certain changes in the dermis.

Distribution.—The labia minora, vestibule, orifice of the urethra and that of the vagina are always affected; the hood of the clitoris sometimes. The skin on the outer surface of the labia majora between the labia majora and folds of the thigh covering the perineum and surrounding the anus is never affected.

Signs.—Kraurosis vulvæ may be divided into two stages. In the first, the muco-cutaneous surface is red, glistening, and shiny, whilst dotted over it here and there, but more particularly on the hymeneal remains round the vaginal orifice, may be seen isolated patches, varying in size from that of a pin's head to half a split-pea, and in colour from that of a bright red to a dull purple. In addition a caruncular condition of the urethral orifice is very often present. In the second or final stage the muco-cutaneous surface becomes pale yellow and glistening, "recalling the colour as well as the aspect of the surface of a fatty liver, particularly that of a goose such as we see exposed for sale in a provision shop." The muco-cutaneous surface is perfectly smooth, all the ridges having become obliterated. The vaginal orifice has contracted, so that perhaps only with difficulty can a digital examination be made; the labia minora and clitoris disappear, the mons veneris atrophies, and the pubic hair becomes brittle and breaks off or falls out. In this disease, though marked shrinkage occurs in the mucous membrane,

yet in contradistinction to leucoplakic vulvitis, owing to the absence of epithelial and subepithelial hypertrophy, the condition may be termed one of retraction with thinning.

Symptoms.—The principal symptoms complained of are those of soreness and pain, together with dysuria and dyspareunia. It is very worthy of notice that whereas in most text-books pruritus is stated to be one of the constant symptoms of kraurosis vulvæ, it is in reality one of the rarest, and that whereas dyspareunia as a symptom is only lightly touched upon, it is in reality the trouble mostly complained of. At least this has been our experience, which also corresponds with that of Thibierge, who remarks in his masterly essay on the subject: "The functional symptoms of kraurosis vulvæ are of an exclusively mechanical order." In the first stage of the disease the parts are extremely sensitive, the passage of urine gives pain, and coitus, for the same reason, is hardly endured or is impossible altogether. In the second stage the soreness may entirely disappear, but owing to the retraction of the vaginal orifice dyspareunia becomes common to both parties.

Pathology.—The microscopical appearances of kraurosis vulvæ are quite unlike those of leucoplakia (figs. 5 and 6). The epithelium is everywhere thinner than normal and the papillæ and interpapillar processes are atrophic and in places have entirely disappeared. The subepithelial connective tissue exhibits marked cellularity, but of a patchy kind. In the areas of a section which correspond to the red patches seen in the surface of the mucous membrane a very large and in places even massive proliferation of plasma cells is seen immediately under a thinned and flattened epithelial layer. Surrounding the plasma-cell proliferation, or partly mixed with it, both lymphocytes and polymorphonuclear leucocytes are seen. The lymphocytes are the more deeply placed, and as the surface is approached the polymorphonuclear leucocytes become increasingly predominant. In the rest of the section simple thinning of the epithelium is manifest, with a certain number of polymorphonuclear leucocytes, lymphocytes, and plasma cells in the connective tissue which underlies it. One of the most striking features, perhaps, is the intercalation of polymorphonuclear leucocytes between the epithelial cells. When stained for elastic tissue the sections show that this is present in all parts save where massive accumulations of plasma cells have occurred. A comparison of these histological appearances with those as described as occurring in leucoplakic vulvitis will show that the pathology of the two diseases is quite different.

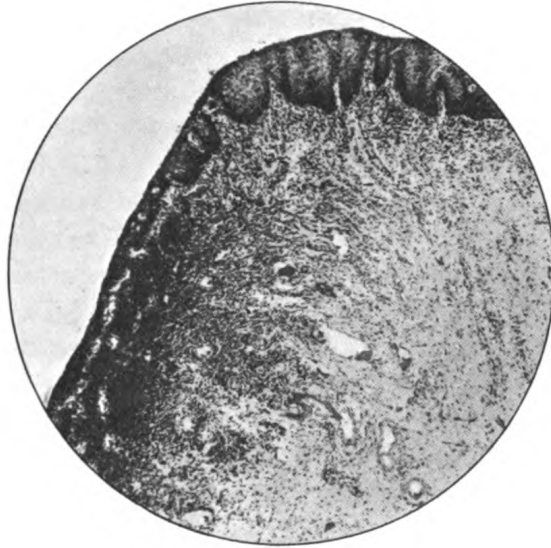


FIG. 5.

Kraurosis vulvæ. The subepithelial tissue is the seat of a massive plasma-cell proliferation, over the surface of which the epithelium is reduced to the thickness of a few cells. Many polynuclear leucocytes are present in the subepithelial tissue and intercalated between the epidermal cells.



FIG. 6.

Kraurosis vulvæ, stained for elastic tissue. There is no loss of elastic fibres except in the areas of massive plasma-cell proliferation.

Ætiology.—Various causes have been assigned to kraurosis vulvæ, such as leucorrhœa, syphilis, sexual abuse, age, and operations upon the genital organs necessitating the removal of the ovaries. Jayle's classification is as follows: (1) Leucoplakia, or white kraurosis (simple or syphilitic); (2) inflammatory, or red kraurosis (follicular or vascular); (2) senile kraurosis; (4) post-operative kraurosis. In our experience only the last two factors have any part in the ætiology. In the light of our investigations this classification must be considerably modified. White kraurosis is undoubtedly a misnomer for leucoplakic vulvitis. Red kraurosis is the first stage of the changes we have described under the heading of this disease. The terms "senile" and "post-operative" kraurosis are merely an indication of the cause and have no place in a classification founded on pathology. Whilst the changes producing kraurosis vulvæ are undoubtedly in a large measure inflammatory, yet there is another factor in its production which is of signal importance. The cases may be divided into three groups: (1) Those in young women associated with sterility; (2) those occurring at or after the menopause; and (3) those occurring after operations involving the removal of both ovaries. It would appear, therefore, that deficiency or absence of some ovarian factor in the economy of the organism plays a large part in the causation of the disease. We know of no instance where kraurosis vulvæ has been associated with carcinoma of the vulva, and the disease has, in our opinion, no relation to it, in this way markedly differing from leucoplakic vulvitis, which, as we will now proceed to show, is very closely connected with cancer in this situation.

RELATIONSHIP BETWEEN LEUCOPLAKIC VULVITIS AND CARCINOMA OF THE VULVA.

The following statistics deal with fifty-eight cases of carcinoma of the vulva and nineteen of leucoplakic vulvitis that have been admitted to the Middlesex Hospital and Chelsea Hospital for Women during the last ten years.

Age.—The average age for carcinoma was 59, that for leucoplakia 51. The oldest patient with carcinoma was 81, and the youngest 40; the oldest with leucoplakia was 70, the youngest 26.

Civil State.—In carcinoma 58·6 were married women, 25·8 widows, and 15·6 single women; whilst in leucoplakic vulvitis 63·2 were married, 21 widows, and 15·7 single.

Parity.—In the carcinomatous cases 48·2 were sterile, and in leucoplakia 47·5. Of those women who were not sterile, the average number of pregnancies in both diseases was seven.

Pruritus and Leucorrhœa.—Thirty-six per cent. of the carcinomatous patients complained of pruritus and 13 per cent. of leucorrhœa, whilst 63 per cent. of the patients suffering from leucoplakic vulvitis complained of pruritus, but none of leucorrhœa.

Ætiology.—As we have already indicated, leucoplakic vulvitis is, in our opinion, an antecedent condition and the cause of carcinoma of the vulva. What is the cause of leucoplakia is a much more difficult question to decide; it has really never been determined. It cannot be due to a leucorrhœa, because in the first place the age does not correspond with the commonest period for leucorrhœal discharges; secondly, the disease never attacks the vagina, which canal should certainly be affected if this were the cause; and, lastly, in our experience of leucoplakic vulvitis, leucorrhœa has been absent. When one comes to consider the civil state of the patients, it is seen that 84·4 of carcinomatous cases and 84·2 of the leucoplakic are, or have been, married; whilst, with respect to parity, both conditions are very similar, about forty-eight of the women being sterile, and of those who were not, seven was the average number of pregnancies in each. Lastly, we see that the average age for both diseases is over 50. Leucoplakic vulvitis is evidently due to some damage to, or interference with, the resisting power of the surface epithelium of the labia; and although coition as a cause of this cannot be insisted upon in every case, it seems to be one of the chief causes when it is remembered that eighty-four of the patients at least have been subjected to coition, and that the age of incidence corresponds fairly closely to the limit of the most active period of this function; whilst the question of parity is of no assistance, since practically the parous and sterile women are in equal numbers. In none of our cases was there any history or signs of antecedent syphilis, nor could evidence of the presence of the spirochæte be obtained by histological methods.

PATHOLOGY OF CARCINOMA OF THE VULVA.

In any case of carcinoma of the vulva we have operated upon, and in every case we have seen, leucoplakic vulvitis has always been present. It has been shown that in leucoplakic vulvitis the maximum hypertrophy of the interpapillar epithelial processes occurs in the second stage—i.e., that in which the cellularity and rarefaction of the subepithelial tissues

are also at their maximum. It is in this stage that the supervention of carcinoma is to be feared. It begins over a variable area as an increasing hypertrophy of the interpapillar epithelial processes, whereby they penetrate more and more deeply into the underlying connective tissue (fig. 7). The connective-tissue papillæ are more than correspondingly elongated, so that they reach much higher than normal, but they are thinned and compressed. It would appear that the growth energy of the epithelial cells becomes confined to those deep down in the interpapillar processes, while the more superficial cells, on the contrary, are of feeble vitality, loosely attached to one another, and actually desquamate before keratinization can occur. Thus the elongated papillar processes of the corium eventually reach the free surface, which now bleeds readily, from punctate points corresponding to their apices. The condition may be likened to an area of skin after a Thiersch graft has been removed. This absence of keratinization and early desquamation of the surface cells of a squamous-celled carcinoma is very striking, and contrasts markedly with the condition found in a papilloma.

The epithelial down-growths, which are at first simply the enlarged pre-existing interpapillar processes of the epidermis, later become branched and tortuous. This development of squamous-celled carcinoma from hypertrophied pre-existent interpapillar processes has been shown by one of us to be a constant feature of the disease wherever its site, for even in positions such as the vaginal cervix, in which normally no papillæ or interpapillar processes exist, a series of such are always formed previous to the onset of squamous-celled carcinoma there, as a result of the chronic cervicitis which is its invariable antecedent. The formation of interpapillar down-growths of epithelium would appear to be an adaptation to relieve lateral growth tension, for it is certain that in all conditions of hypertrophy of the lower strata of an epidermis they either appear *de novo* (cp. chronic cervicitis), or, if normally existent, become much exaggerated (cp. the skin). Further, it would appear that so long as the growth energy of the epithelial cells is evenly distributed along the length of the epidermis, enormous hypertrophy may take place without malignancy. But that when the growth energy becomes concentrated at a series of points corresponding to each interpapillar process, a want of co-ordination is established which at once makes the condition dangerous.

The relation between the malignant epithelial ingrowth and the altered state of the subepithelial tissues is an important subject. We have in leucoplakic vulvitis two sets of phenomena going on simultaneously—namely, those in the epithelium and those in the connective

tissue. In both of them an accession of cellular energy takes place, which then declines and finally gives place to a state of cellular activity and reproduction much below the normal. But a study of the earliest stages of the disease shows that the activity of the connective-tissue cells is that first awakened, the growth of the epithelial cells initially being actually inhibited. Thus it comes about that a profoundly altered state of the subepithelial tissue anticipates the occurrence of epithelial hypertrophy.

Further, it would appear that a condition of rarefaction and cellularity of the subepithelial tissue is an important factor, influencing epithelial ingrowth. For in the earliest phases of carcinoma of the vulva in which the epithelial ingrowth is only as yet foreshadowed, the connective-tissue-cell proliferation around this area is already typical of that seen in the developed disease. The disappearance of elastic tissue in the area of a

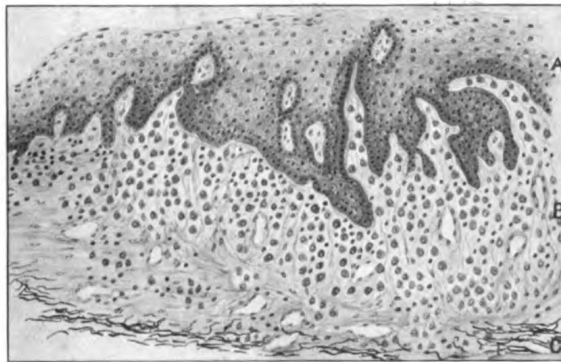


FIG. 7.

The earliest stage of squamous-celled carcinoma of the vulva. It begins as an increasing hypertrophy of the interpapillar epithelial processes, whereby they penetrate more and more deeply into an underlying connective tissue which is already crowded with plasma and other inflammatory cells, and from which all elastic fibres have previously disappeared. **A**, thickened epithelium with large interpapillar processes. **B**, subepithelial zone, the seat of diffuse rarefaction due to inflammatory cell proliferation and disappearance of collagen and elastin. **C**, normal tissue with elastic fibres. (Camera lucida drawing.)

commencing carcinoma is very striking (fig. 8). On the other hand, there is evidence to show that an excess of these fibres probably acts as a check to epithelial ingrowth. In certain very chronic types of the disease, in which the subepithelial changes never reach a high degree of cellularity, a great excess of elastic tissue may be found. In the later stages of this condition considerable epithelial hypertrophy occurs, but without those

long interpapillar downgrowths which are the first indications of approaching malignancy (fig. 9). Further, the more benign papillomatous type of carcinoma of the vulva shows less destruction of elastic tissue than the more malignant ulcerative form. Dense sclerosis of the white fibrous elements also probably checks epithelial ingrowth, and, as this progresses in the later phases of leucoplakic vulvitis, the tendency to carcinoma becomes increasingly diminished.

Of the cellular elements in the connective-tissue the plasma cells are those which most characterize the second stage of leucoplakic vulvitis, and these are also the predominant tissue cells in carcinoma of the vulva. They bear this relation to the initiation of epithelial ingrowth—namely,



FIG. 8.

The tissues at the edge of a squamous-celled carcinoma of the vulva. The epithelium is hypertrophic and is growing down into the de-elasticized zone, **A**; below this are seen normal elastic fibres, **B**. The edge of the carcinoma is just beyond the left of the field; masses of plasma cells are seen in that direction.

that they are largely responsible for the decollagenization of the sub-epithelial tissue which occurs in the antecedent leucoplakic vulvitis. Though changes in the adjoining connective tissue bear some very close relation to the cause of epithelial ingrowth, yet malignancy having been established, the further spread of the tumour is independent of such assistance. For one of us has shown that the condition of the connective tissues surrounding an advanced inrunner of carcinoma cells is one of passivity (fig. 10), and that the same is true of the tissue



FIG. 9.

Very chronic type of leucoplakic vulvitis with excess of elastic fibres in the connective tissue. The epithelium is hypertrophic, but the interpapillar epithelial processes are smaller than normal.



FIG. 10.

Inrunners of carcinoma cells in advanced carcinoma of the vulva, showing the passivity of the invaded tissue.

surrounding permeated lymphatics (fig. 11) and metastatic growths (fig. 12),¹ excepting those in lymphatic glands, which are an exception to this rule.

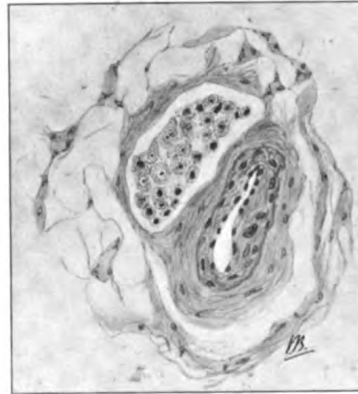


FIG. 11.

A small lymphatic channel containing a mass of carcinoma cells. The tissues of the wall of the channel and those outside it are passive. (Camera lucida drawing.)

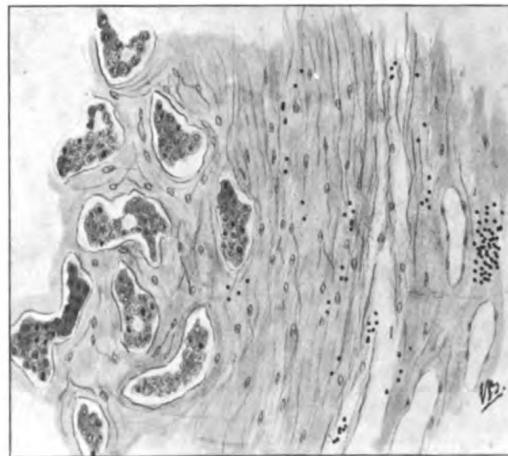


FIG. 12.

Portion of a secondary subcutaneous nodule of carcinoma, showing the passivity of the surrounding tissue. (Camera lucida drawing.)

¹ Victor Bonney, loc. cit. The fact concerning lymphatic channels permeated by carcinoma was first stated by Sampson Handley, "Hunterian Lecture," Royal College of Surgeons, 1905, i, p. 984.

MULTIPLE CARCINOMATA OF THE VULVA.

Multiple carcinomata are more frequently met with in the vulva than any of the other common sites of this disease. This occurrence has been interpreted by some as an evidence of auto-inoculation. The cases that have been interpreted in the manner mentioned may be divided into two classes: pseudo-multiple growths and true multiple growths.

(A) Pseudo-Multiple Growths.

Many growths which at first sight appear to be double are in reality bridle-shaped, a thin tract of carcinomatous tissue uniting them across the middle line, usually just under the clitoris. When a growth crosses the middle line it rarely does so directly by way of the vestibule. This is due to the fact that the vestibula mucosa usually escapes the antecedent vulvitis—that is to say, it is not in a pre-carcinomatous state. Ribbert stated, and we confirm his view, that the surface spread of a carcinoma is at first by successive carcinomatous conversion of the epithelium covering the pre-carcinomatous area. When this is exhausted further growth continues by division of the cells already formed, but there is no conversion of normal epithelium into carcinoma, the former simply undergoing a pressure destruction owing to the infiltration of the subepithelial tissues by the cells of the growth.

(B) True Multiple Growths.

We have seen four such. In three out of the four they occupied the same side of the vulva, and in none of them do we think that there was any reason to believe that they arose by auto-inoculation. In all of them the vulva was leucoplakic, and the double growths were separated by a tract of mucosa that showed the typical changes belonging to that condition. Each separate growth was an entity to itself, starting in its own set of hypertrophic interpapillar processes, and in three of the cases contact between the two tumours was, from their position, impossible. Moreover, there was no microscopical evidence of any new element having been introduced either from the surface or from the subcutaneous tissue. Such multiple growths, therefore, should neither be attributed to auto-inoculation from the surface nor to metastatic deposit via the subcutaneous tissue. Microscopically it is quite common to find dual, treble, or even quadruple points of beginning carcinoma. In fact, seeing that every down-growth begins as a hypertrophic interpapillar epithelial

process, the growth may be said always to begin at multiple points. The rarity of multiple primary growths of the vulva, in spite of the large pre - carcinomatous area existent, suggests the possibility that the epithelial ingrowth at the point of the carcinoma may act as a safety valve to the increased lateral growth tension that undoubtedly exists throughout the rest of the leucoplakic epidermis and relieves the latter of its tendency to ingrowth.

LOCAL RECURRENCES OF THE GROWTH.

There are two types of local recurrence. The first is that where the original growth has been inefficiently removed and recurrence takes place

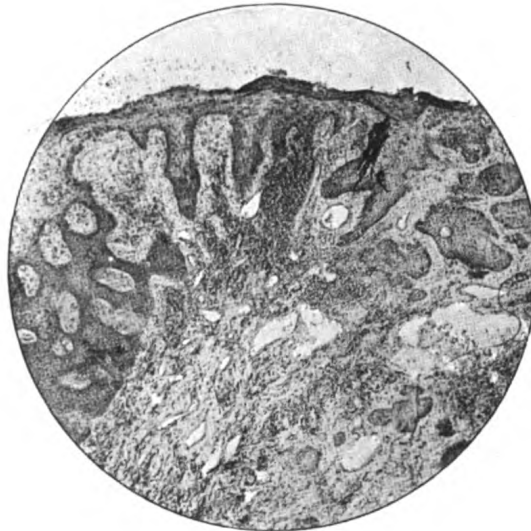


FIG. 13.

Portion of a nodule of carcinoma that occurred on a recurrent patch of leucoplakic vulvitis, left after imperfect primary excision. The tissue on the right is carcinomatous; that on the left leucoplakic.

in the scar a short time afterwards. We have met with none such. The second is of a different nature, and we have met with examples of this after excision of the vulva for leucoplakic vulvitis. It is due to a separate cancer originating *de novo* from a portion of the pre-carcinomatous area that has escaped removal (fig. 13). It is here to be noted that, unless the lines of incision for removal of the vulva for leucoplakic vulvitis are so planned as to entirely lie without the diseased area, the leucoplakic

condition returns in the scar. These recrudescences in a portion of leucoplakic epithelium left behind at the original operation are in no way to be confounded with secondary metastatic nodules. Such nodules are subepidermic in origin, whilst the recrudescence is obviously arising from a certain group of hypertrophied interpapillar processes belonging to an area the whole of which is leucoplakic and pre-carcinomatous (fig. 10).

GLANDULAR METASTASES.

Glandular involvement occurs late as compared with squamous-celled carcinoma in other sites. The inner set of horizontal inguinal glands are those first affected on one or both sides. When affected they rapidly enlarge, and subsequently often break down. Sections cut from them at

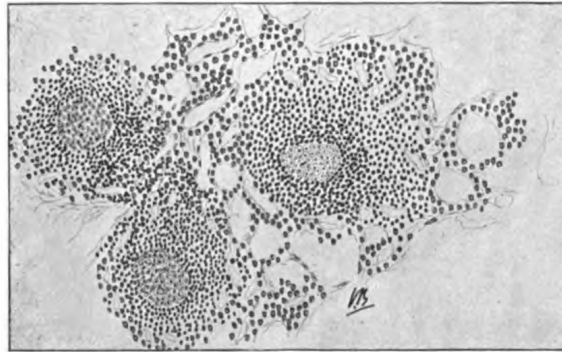


FIG. 14.

Section of a lymphatic gland, enlarged but not carcinomatous; the trabecular elements are the seat of a plasma-cell proliferation. (Camera lucida drawing.)

this period may fail to demonstrate the nature of the change, so complete is the destruction of the cells that has taken place. All enlarged glands are not, however, carcinomatous. It has been shown by one of us¹ that lymphatic glands prior to their invasion by carcinoma cells are enlarged by a plasma-cell proliferation affecting the trabecular elements, together with a great hypertrophy of the germinal areas and an increase in number of the lymphocytes derived from them (fig. 14). Glands thus affected may attain a large size, mimicking true carcinomatous

¹ Victor Bonney, *ibid.*

enlargement so closely that microscopical investigation is alone competent to decide its nature.

TREATMENT.

We have tried a large number of remedies for the pruritus in leucoplakic vulvitis, and have found that most of them have failed to give relief. Of these, the most successful have been zymocide lotion, resinol ointment, and the X-rays. But in view of our pathological findings we advise that where these fail the affected area should be excised, and, since we have shown that a permanent change of character has taken place in all the epithelial cells over the affected area, the excision should be a wide one, lest the disease recur again in the scar, as has happened to us in two of our cases. The treatment for kraurosis vulvæ is to dissect out the painful parts complained of and enlarge the vulval orifice. In carcinoma the whole vulva should of course be removed, together with the inguinal glands on both sides, whether enlarged or not.

DISCUSSION.

The PRESIDENT (Dr. Macnaughton-Jones) said that this was a most valuable gynæcological contribution, and he congratulated not only the authors but this Section on its having been read in it. It broke completely new ground, and threw new light on a condition about which much confusion existed. To-night they had had a clear differentiation, most beautifully illustrated, between two distinct pathological states which previously had been regarded as different stages of the same disease. No such clear distinction had by any authority yet been drawn, the most general view, held up to quite recently, being that it was a chronic inflammatory condition. He would suggest that, as the time was short for discussion, they should deal with it from the standpoint of the individual experience of each Fellow as to the ætiology, clinical aspects, and pathology of the disease or its passage into malignancy.

Dr. ARTHUR E. GILES congratulated Dr. Berkeley and Dr. Bonney on having brought forward such a valuable paper, which shed a much-needed light on a hitherto obscure corner of gynæcological pathology. With regard to the ætiology of kraurosis of the vulva, there was no evidence in any case that had come under his notice of any history either of syphilis or of tuberculosis; but he had regarded the condition as an atrophic one, incidental to the general atrophy that affected the pelvic organs at the time of the menopause; but the

question still remained unsolved why such atrophy should affect some patients and not others. The specimen that he had shown earlier was from a patient in whom the clinical signs were those of kraurosis rather than leucoplakia; but carcinoma might follow kraurosis as well as leucoplakia.

Dr. AMAND ROUTH agreed that the authors had brought forward very strong arguments in favour of their contentions that leucoplakia and kraurosis vulvæ were essentially distinct conditions, capable of easy clinical differentiation. If their contention were correct, all modern text-books were wrong, for, without exception, they described the two conditions under one name—kraurosis vulvæ—grouped the symptoms of pruritus and dyspareunia as occurring in different stages of the same disease, and made no distinction as to the respective tendency towards carcinoma. The authors, on the other hand, had so dogmatically stated the differences that their distinction between the two diseases was at once obvious. According to the authors, in leucoplakia, pruritus was the main, often the only, symptom; neither the vestibule nor the urethral orifice was ever attacked, but the disease tended to spread over the inner thighs, perineum, and anus, and there was no tendency to contraction of the vaginal orifice. Epithelioma of the vulva very often followed this condition. In kraurosis vulvæ, on the other hand, there was no pruritus, and dyspareunia was the main symptom. The vestibule and urethral orifice were always attacked, the thighs, perineum, and anal region never. The vaginal orifice became always contracted, and carcinoma never developed on the vulva in kraurosis. If it really was the fact that these two diseases were entirely distinct, and not, as had been previously thought, merely early and advanced stages of the same condition, he felt that sometimes the two diseases must coexist and overlap, and he described a case in point:—

Mrs. R., aged 55, whom he had seen in November, 1907, with the late Dr. Picard, of St. John's Wood, had had a leucoplakial condition of the vulva with marked pruritus for two years, but during the last few months had suffered from soreness near the left labium minus, and from dyspareunia owing to narrowing of vaginal orifice. On examination this seemed a typical case of leucoplakia passing on to kraurosis. The elevated plaque near the left labium minus continued to enlarge, and eventually Dr. Routh removed it widely, and Dr. Lockyer reported that it was an epithelioma which had developed from a primary papilloma.

As regards treatment, he had found that the continued application of equal parts of unguentum hydrargyrii and unguentum zinci or unguentum plumbi acetatis almost always relieved the pruritus and made the tissues softer and pinker, but the condition was apt to recur. He had not seen any case with a definite history of syphilis.

Dr. EDEN said that the pathology of the morbid conditions of the vulva had been in a state of confusion for so long that we must welcome any successful attempt to clear up the subject. It appeared to him that the authors deserved great credit for the valuable histological work they had done in respect of two of these conditions. There was nothing inherently improbable in the idea that leucoplakia was a condition antecedent to epithelioma, for this sequence

had for long been recognized as occurring in the tongue. But he could hardly follow the authors in their recommendation that all patches of leucoplakia should be treated by free excision, for there was no doubt that, while leucoplakia was common, epithelioma was very rare, and the malignant transformation described in the paper must be quite an uncommon occurrence.

The PRESIDENT said that the last edition (1904) of his text-book was open to the criticism of Dr. Routh, for though the views of Breisky, Sanger, Orthmann, A. Martin, Jung, and Heller were epitomized in it, there was no mention of the term leucoplakia. The two conditions were dealt with under the one head of kraurosis vulvæ, in which the characteristic aphthous patches and the atrophic areas of "wash-leather" appearance were present. In regard to its ætiology, he had never been able to associate the cases he had seen with the history of syphilis, nor were they tuberculous. In two very well marked cases there were endometritic discharges (not gonorrhœal), and in one of the worst he had seen a cervical polypus and anal fissure complicated the condition. In two cases there were also hæmorrhoids with rectal fissure present, and in one a deep fissure of the fourchette. His experience would lead him to regard discharge, both uterine and vaginal, and senility as important factors in its ætiology. He was fortunate, for no patient he had treated had he known to suffer from carcinoma subsequently. As to treatment, he believed in free extirpation of the "wash-leather" areas, though not to the exclusion of other therapeutic measures, of which he considered the application of pure carbolic acid to the aphthous regions and the free use of 5 per cent. nitrate of silver solution to be the most effectual. He had tried soakage of the parts with adrenalin with benefit. Lanolated ichthyol ointment, in combination with the yellow oxide of mercury and emollient baths, followed by the application of the nitrate of silver, was also very useful. He quoted two extreme cases which were completely cured by the combination of excision and transplantation of adjacent sound skin aided by the remedies mentioned. He believed that whilst nothing save the extreme radical step advanced by the authors would suffice in certain cases, still there were others which could be cured without. The condition was one that rendered the woman's life miserable, and her suffering was extreme. The intolerable pruritus only ceased when there was final and complete atrophy.

Dr. COMYNS BERKELEY thanked the President and Fellows for the kind way in which they had received the paper. He noticed most of the speakers had directed their remarks to two points: (1) the ease or otherwise of diagnosing the separate diseases of leucoplakia and kraurosis, and (2) on the advisability of following the treatment advocated for leucoplakia vulvitis. He (Dr. Berkeley) said that the two diseases were very easy to diagnose, and it was in fact their marked difference on inspection that had first drawn the authors' attention particularly to the subject. He noted that some of the Fellows present agreed that when they came to cast their minds back on the cases they had seen, there was a marked difference between those which they had

always considered to be the same disease. With regard to treatment, Dr. Bonney and himself were convinced that the proper treatment of leucoplakia was to excise the diseased area. The speaker had had probably somewhat an unusual experience, since he had operated upon eighteen cases of carcinoma of the vulva all complicated with leucoplakia, and at least three of which had developed carcinoma after a prolonged treatment for the leucoplakia. He did not see why leucoplakia and kraurosis should not occur in the same patient, but he had never seen a case.

A Large, Solid Ovarian Tumour. ? Adeno-carcinoma.

By ARTHUR E. GILES, M.D.

THE patient from whom this tumour was removed was a widow, aged 60. She was under the care of Dr. Wilfred Thomas. The patient had known of the existence of an abdominal tumour for about sixteen years. Ten years ago Dr. Thomas sent her to see a well-known specialist with a view to the removal of the tumour; a diagnosis of uterine fibroids was made and she was advised to wait, on the ground that the menopause might bring about a diminution of her tumour. Instead of diminishing, however, the tumour continued to increase, and when I saw her in September of this year it occupied practically the whole of the abdomen, extending up to the costal margins, and causing a great deal of discomfort and inconvenience by its mechanical effects. The patient walked with difficulty and became very short of breath on exertion. The tumour felt very hard and slightly irregular on the surface. I made a diagnosis of uterine fibroids of the pedunculated subperitoneal type, and advised operation. She was admitted to the Prince of Wales's General Hospital, Tottenham, and the operation was performed on September 23.

The tumour proved to be ovarian; there were no adhesions, and the operation was so simple that it took only twenty minutes from start to finish, most of this time being occupied in sewing up the abdominal wound, which had to be of a great length, extending from the ensiform cartilage to the pubes, in order to allow for the extraction of the tumour. The patient made a smooth recovery, and returned home on the twenty-second day.

The tumour is almost entirely solid and weighed 18 lb.; a small portion presents the characteristics of a multilocular cyst. The result of a microscopical examination, conducted at Dr. Eastes' laboratory, revealed the existence of adeno-carcinoma.

The long history makes it probable that the growth was not malignant to start with; it probably began as a fibro-adenoma, the malignant feature being of a later development. The case illustrates the undesirability of advising patients to wait until the menopause when uterine fibroids are present, because, apart from the drawbacks of this course when the diagnosis is correct, there are cases in which it is impossible to differentiate between pedunculated fibroids and solid ovarian tumours; and inasmuch as the latter are not infrequently malignant, it is obvious that delay may involve serious risk to the patient.

In three other cases in which I operated for supposed uterine fibroids the tumours were partly ovarian and carcinomatous, uterine fibroids being also present.

DISCUSSION.

Dr. HERBERT SPENCER said that the dense, hard, non-degenerated character of the growth, notwithstanding its long duration and great size, pointed to its non-malignant nature. The growth was, he thought, an adeno-fibroma, and closely resembled in structure one shown by the President as an "anomalous ovarian tumour," published in vol. xl of the *Obstetrical Transactions*, and illustrated by a plate in the opposite page of the same volume, which might escape notice, as there was no description upon the plate nor reference to the report of the Pathology Committee, which appeared at p. 213. The speaker had excised a similar growth of small size from an ovary.

Dr. AMAND ROUTH alluded to the absence of ascites in this case. Ascites was present in 40 per cent. of cases of all solid tumours of the ovary, and almost invariably present in malignant solid ovarian tumours, whether sarcomatous or carcinomatous.

Dr. LOCKYER said that it had been correctly assumed that he knew nothing about the clinical history of the case when he described Dr. Giles's specimen as an adeno-carcinoma. Dr. Lockyer found masses of proliferating epithelial cells occupying and filling up alveolar spaces in a dense fibrous stroma. No simple tubular spaces, lined by columnar epithelium, were present; in fact, the tumour was histologically malignant, in spite of its clinical history.

Dr. BRIGGS, from his experience with similar unilateral growths and from the histological characters shown in the section, was of opinion that the growth was non-malignant. The more actively cellular adenomata of the ovary may present insuperable histological difficulties in diagnosis.

The PRESIDENT (Dr. H. Macnaughton-Jones) said that the ovarian tumour referred to by Dr. Spencer was one that complicated a uterine myoma and produced symptoms of morbus coxarius through pressure on the sciatic nerve. It was of quite a different character to this specimen shown by Dr. Giles, and, though at the time thought by some to be malignant, was subsequently by a Pathological Committee proved to be an adeno-fibroma. The most interesting case of adeno-carcinoma of the ovaries he had ever seen was one in a patient who had passed the menopause and in whom no malignancy was suspected; both ovaries were found affected and enlarged to the size of cocoanuts, soft and semi-cystic; the tubes were free of disease, and there were two pedunculated myomata of the uterus. The patient lived for some six months after operation, dying, he believed, from recurrence of carcinoma in the bowel. The presence of ascitic fluid, though more frequent when there was malignancy, was by no means universal in such cases. His experience of Dr. Lockyer's accuracy was such that he felt inclined to regard this tumour as malignant until the contrary was proved.

Mr. GLENDINING remarked that he had recently cut and examined histologically a very similar case, and found there a point in favour of its malignant nature—permeation of the lymphatics of the mesosalpinx. The presence of growth in the lymphatics seemed to him to decide the character of the growth.

Dr. DARWALL SMITH mentioned, as bearing on the question of the malignancy of these tumours, that he had brought a somewhat similar tumour before the Section recently, and that in his case the patient was now perfectly well and without any sign of recurrence more than two years after operation; further, that the tumour had been known to have been present and to have remained approximately unaltered in size for eighteen months before operation.

Report of Pathology Committee on Dr. Giles's Specimen.—A fibro-adenoma of the ovary of unusual character, with marked proliferation of the epithelial cells, which, however, are encapsuled and do not present definite malignant characters. Dr. W. S. A. Griffith, Dr. J. S. Fairbairn, Dr. T. G. Stevens, Dr. G. Blacker, Dr. H. Williamson, Dr. A. E. Giles, and Dr. C. Lockyer wish it to be recorded that in their opinion the groups of epithelial cells found in the alveolar spaces in the fibrous stroma are malignant.

A Case of Kraurosis of the Vulva, with Commencing Carcinoma.

By ARTHUR E. GILES, M.D.

THE patient, a widow, aged 79, was under the care of Dr. W. H. Briand. She had suffered from troublesome irritation of the vulva, with pain on sitting down, on walking, and on passing water. Examination showed a condition of kraurosis of the vulva of an advanced type. Just within the vagina at the vulvo-vaginal margin, on the left side, there was a hard nodule the size of a pea, which felt suspiciously like a malignant growth. I advised removal of the whole of the affected area of the vulva and of a portion of the vagina involved by the growth. This was carried out, and a microscopical examination of the nodule showed that it was carcinoma.

It has long been recognized that kraurosis of the vulva may be a precursor of carcinoma; I have met with about a score of cases of kraurosis, and this is the second in which malignant disease had supervened, so that the liability to this complication may be looked upon as a definite one. When kraurosis is so troublesome as to necessitate operation, the procedure that I have usually adopted is as follows: An incision is carried round the vulva just outside the affected area, a second incision skirts the margin of the vagina and vulva, passing in front of the urethra in its anterior portion; the whole area between these two incisions, involving the labia minora and majora and the clitoris, is removed; the cut edges are brought together, the resulting scar being triradiate; the junction of the three limbs of the scar is just in front of the urethra. In this case the procedure was modified by carrying the inner incision higher up in the vagina.

Fibromyoma and Pregnancy, Thrombosis of Veins and Necrobiosis of the Tumour.

By T. G. STEVENS, M.D.

THE patient from whom this specimen was removed was aged 39, and had had five children during the previous three years. She was first seen on April 1, 1909, and gave the following history: Menstruation

had been regular until January, 1909; after that date there was complete amenorrhœa. There was no retching or vomiting, the breasts were enlarged and tender, and on examination the uterus was found to be enlarged and reached to the level of the umbilicus. It was somewhat soft, but had not the consistence of a normal pregnancy, and no foetal parts could be felt through the abdominal wall. *Per vaginam* the posterior surface of the uterus was softer than the anterior, and it was thought that some part of a foetus could be felt. The provisional diagnosis of pregnancy was made, but whether normal or pathological could not be determined.

The patient was again seen on May 11, when the tumour had enlarged and reached 1 in. above the level of the umbilicus. It was soft and uniform in consistence in front; no foetal parts could be felt through the abdominal wall nor *per vaginam*. Nevertheless, the tumour was softer and felt more like a normal pregnant uterus through the posterior vaginal fornix. In consultation with Dr. Eden the diagnosis of pregnancy plus a tumour, probably fibromyoma, was made. At the same time it was not at all clear that the tumour was a fibroid, and an ovarian cyst or malignant tumour could not be excluded. An exploratory operation was considered advisable, owing to this diagnostic difficulty. A few days later there was a sharp attack of acute pain over the tumour and fever lasting three days. This strengthened the determination of the patient to submit to the operation. At the operation on May 25 the tumour was found to be a softening fibroid of the anterior uterine wall, so placed that it raised the peritoneal reflection in front 4 in. The pregnant uterus was behind the tumour and incarcerated in the pelvis, making it appear very improbable that the pregnant uterus could rise up into the abdomen. Further, the fibroid was so attached that it would probably cause obstruction to delivery if left alone. On these grounds the whole tumour was removed by supravaginal amputation. The patient made an uninterrupted recovery. The tumour on section after preservation whole in 10 per cent. formalin in normal saline solution presented a remarkable appearance. All the veins in the capsule were thrombosed, the thrombi being shown to be old by their microscopic appearances. Between the capsule and the fibroid was a layer of coagulated fluid exudate, whose thickness varied from $\frac{1}{8}$ in. to $1\frac{1}{2}$ in., the thickest part being at the upper end of the tumour, corresponding with the seat of the pain referred to. This exudate must be presumed to be an acute condition, the result of the universal thrombosis of the veins in the capsule. The tumour itself showed many large

areas of necrobiosis, with their usual red colour and histological staining reactions. It has been before suggested that thrombosis of veins in the capsule is a cause of necrobiosis, but this is such an extreme condition that it was deemed worthy of notice. No cultures were made from the tumour, but no organisms could be found in sections stained by Gram's method. There is no satisfactory cause to be suggested for this widespread thrombosis of vessels.

DISCUSSION.

Miss ALDRICH-BLAKE had been interested to hear this account of a case of necrobiosis occurring in the earlier months of pregnancy, as she had had a somewhat similar case under her care a few years ago. The case was that of a married lady, aged about 34, whom she saw in the fifth month of her first pregnancy. The patient had been seen in the third month by her doctor, Dr. Alice Corthorn, when it was noted that the size of the uterus suggested a pregnancy considerably more advanced than would correspond with the length of the amenorrhœa. In the course of the fifth month, after sitting a long time in a very cold hall in a provincial town, patient was seized with a severe pain in the right lower abdomen. The pain continued and the part was very tender, but she managed to return to London, and was placed at once in a nursing home. Temperature rose to 102° F. and 103° F., and the pain was so severe that morphia was given. A prominence, the size of a closed fist, could be felt near the right cornu of the uterus; it appeared closely incorporated with the uterus, but extreme tenderness made examination difficult. The diagnosis lay between a fibroid undergoing some degeneration and a cyst with torsion of pedicle. In favour of the former was the fact that the uterus had been felt to be abnormally large at an early date of the pregnancy. Exploration was decided on: a large interstitial fibroid was found, one half of which formed a prominence, the other half being buried in the uterine wall. An incision about 4 in. long was made over the prominence, the fibroid was shelled out, and the cavity closed by three layers of catgut suture, with a separate silk suture for the surface peritoneum. The patient was delivered of a healthy child at full term, and has since had a second normal pregnancy. Miss Aldrich-Blake could not state the condition of the capsule with respect to thrombosis in her case, as none was removed; but the tumour was throughout of a bright purplish red colour, and quite unlike an ordinary fibroid in appearance. She did not mean to suggest that in the case under discussion the tumour could have been enucleated, but it was of interest to her to hear of a case with so similar a history, as many recorded ones occurred after parturition.

In reply to Dr. Aldrich-Blake, Dr. STEVENS said that he considered myomectomy would have been impossible in this case.

Adenomyoma of the Vaginal Wall.

By T. G. STEVENS, M.D.

THE patient from whom this specimen was removed was single, aged 32, and was seen in consultation, complaining of menorrhagia and metrorrhagia. She had been curetted nine years previously for excessive losses at the periods. There was no history of any illness bearing on the condition, and the uterus on examination showed only trifling enlargement. The left ovary was a little larger than the right, but did not suggest any pathological condition. There were, however, two little tumours to be felt—one in the middle line of the anterior vaginal wall, an inch away from the cervix, the other $1\frac{1}{4}$ in. away from the cervix in the postero-lateral vaginal wall. By the speculum the first, which measured about $\frac{1}{2}$ in. in diameter, could be seen to be cystic and felt like a cyst. The posterior one, however, was hard and felt like a solid tumour; it consisted of two small structures joined by a cord, each portion measuring about $\frac{1}{4}$ in. in diameter. Curettage was performed for the menstrual anomalies, and the endometrium was found to be thickened and in a condition, histologically, of chronic hypertrophic endometritis. The two little growths were removed, the posterior with some difficulty, as it was not attached to the mucous membrane, but lay in the connective tissue between the vagina and rectum. The microscopic characters of these tumours proved interesting. The anterior one consisted of several small cystic cavities lined by columnar epithelium and surrounded by connective tissue and occasional bands of smooth muscle. Side by side with these cysts were occasional tubules lined by cubical epithelium and surrounded by concentric layers of connective tissue, exactly like those seen around the tubules of the parovarium. Dr. Stevens suggested and believed that these cysts and tubules represented displaced Wolffian tubules, and showed a section of some normal Wolffian tubules from an infant's parovarium in support of his contention.

The solid posterior tumour consisted of two portions, with identical characters histologically. They were composed of interlacing bands of smooth muscle tissue, enclosing here and there gland tubules. These were surrounded by delicate connective tissue like that of the endometrium, and were lined by low columnar epithelium. In other words,

the tumours had the usual characters of the diffuse adenomyoma of the uterus. The position of the tumour, absolutely unconnected with the uterus as far as could be ascertained, suggested that its origin must be different from that of the diffuse adenomyoma of the uterus. The original view of von Recklinghausen that these tumours were Wolffian in origin, and not Müllerian, is conclusively negated by Cullen's researches, who believes that even adenomyoma of the round ligament is strictly Müllerian in origin. From the presence of Wolffian tubules in the anterior vaginal wall, Dr. Stevens contended that a Wolffian origin was more than likely for the small adenomyoma, especially as it was unconnected with the uterus and nowhere near the round ligament, ovarian ligament, or utero-sacral ligament.

Rupture of the Uterus treated by Suture of the Rent per Vaginem and Drainage.

By HERBERT J. PATERSON, F.R.C.S.

IN view of the recent discussion on the treatment of rupture of the uterus, the notes of the following case, which has recently been under my care in the London Temperance Hospital, may prove of interest.

E. P., aged 25. Three children previously. After the birth of her third child, eighteen months ago, the patient was laid up in the infirmary for fourteen weeks, and some operation for "womb trouble" (? curetting) was performed. There was nothing unusual in connexion with the two previous confinements.

The history of her recent confinement is as follows: The waters broke on August 11, 1909. Dr. Mulloy saw the patient on August 13. The cervix was not dilated, there were no labour pains, and the cord was prolapsed and pulseless, so nothing was done. Dr. Mulloy was called again at 10 p.m. on the night of August 14. The patient was then having labour pains, but there was no dilatation of the cervix, and the cord was prolapsed and pulseless. Under chloroform the cervix was slowly dilated until the knuckles of the open hand could be passed through the internal os. The right foot and hand presented with the pulseless cord between them. Delivery was effected without difficulty by traction on the foot, the back of the child being rotated towards the pubes, and flexion of the after-coming head secured by introducing a

finger into the child's mouth. No instruments were used. The placenta was expressed with ease. On introducing the hand into the vagina, it passed into the abdominal cavity. Dr. Mulloy, realizing the gravity of the patient's condition, at once sent her up to the hospital.

Condition on admission (note by Dr. Russell Square): There was some bleeding from the vagina, and the patient was very collapsed, sunken round the eyes, and extremely restless. Extremities and face cold and damp with perspiration, pulse very small, soft and uncountable. Some tenderness over the abdomen, increased by gentle pressure with the hand. Some dullness in the flanks. When I saw the patient about 3 a.m. she was extremely collapsed, although somewhat better than on admission, pulse very feeble, rate about 140. The abdomen was somewhat distended, and there was some dullness in the flanks. The vagina was full of blood-clot, and a coil of intestine was felt protruding through a large rent in the vaginal vault. Ether by the open method was given. A loop of large intestine (apparently sigmoid) was prolapsed through an extensive rent in the posterior vaginal wall, through which the fist could be easily passed into the abdominal cavity. The left parametrium was involved in the tear, and there was free arterial hæmorrhage from two large vessels. These were secured by ligature. The large intestine was pushed back into the abdominal cavity, and a considerable quantity of blood was removed from Douglas' pouch. Further examination showed that the cervix was completely torn through, the tear extending upwards and involving the uterus for a distance of at least 4 in., but as the uterus appeared to be firmly retracted no attempt was made to define accurately the upper limit of the tear. The torn edges of the cervix and lower part of the uterus were approximated by three stout catgut sutures. The torn edges of the peritoneum and vaginal wall were seized with forceps, and a purse-string suture was passed right round the tear, the suture being passed alternately through the vaginal wall and peritoneum. A large rubber tube was then inserted into Douglas' pouch, and the purse-string suture tied tightly round it. The patient was much collapsed at the end of the operation, which lasted under half an hour, but gradually rallied. Continuous saline proctoclysis was maintained for sixty hours, and the patient made a good recovery, although her temperature varied between 99° F. and 101° F. for the first twelve days after admission. The tube was removed on the sixth day.

On September 8 the patient was examined under ether. Uterus not felt above pelvic brim. Cervix posterior in pelvis, texture firm, canal closed. Vagina narrowed posteriorly. In posterior fornix a roughened

area about 1 in. in diameter. *Per speculum* was seen a granulating area rather larger than a shilling-piece immediately behind the cervix. Bimanually the uterus was freely movable. Sound passed, concavity backwards, 4 in. *Per rectum* was felt a slight thickening on posterior wall of uterus extending more than halfway up towards the left cornu. The pelvis was not contracted.

So far as I am aware, the method of vaginal suture and drainage was not adopted in any of the recorded cases of ruptured uterus. It appears to me that those who advocate the treatment of this accident by hysterectomy lay too much stress on the difficulty of controlling hæmorrhage. I think that Nature's ability to deal with internal hæmorrhage is often underestimated. A perusal of the recorded cases leads one to think that in some instances recovery ensued *not* because of, but in spite of, hysterectomy. To perform hysterectomy if suture be practicable seems to me to be an unsurgical procedure. As regards the case here recorded, I am strongly of the opinion that the patient was not in a fit condition for hysterectomy, and that this method of treatment would most probably have led to a fatal result.

Obstetrical and Gynæcological Section.

December 9, 1909.

Dr. H. MACNAUGHTON-JONES, President of the Section, in the Chair.

Abdominal Hysterectomy, sixty-three hours after Labour, for a Necrosed and Suppurating Subperitoneal Fibroid.

By PHILIP D. TURNER, M.D.

MRS. S., aged 32, married thirteen months, nullipara, consulted me March 22, 1909, on account of a hard lump which she had noticed in the left side of her abdomen for about a fortnight. Her last menstrual period ended on January 18, and the ordinary symptoms of pregnancy were present. I found a firm, elongated tumour lying on the left side of the uterus, which was about two months pregnant. The tumour rose 5 in. above the pubes, and was freely movable but connected to the uterus. On a subsequent examination a few days later it was lying in front of the uterus. I considered it to be a pedunculated fibroid, and took her into hospital with the intention of removing it.

On April 8 I opened the abdomen and found that the fibroid was attached to the uterus by a very broad base, and I did not consider that it could be removed without terminating the pregnancy or without considerable risk. As its position on the left angle of the fundus was not likely to allow it to interfere with the course of pregnancy or labour, I decided to let the case go on to term, and perform a myomectomy later on.

As the patient returned to her home at Southampton I did not see her again till early in October, when she returned to Ryde, that I might attend her in her confinement. Till about the seventh month the pregnancy proceeded in a normal manner, but from that time

she had pain in the region of the fibroid after any exercise; but it gave her no trouble so long as she rested. The tumour was situated in the left hypochondrium and partially concealed by the thorax, but it felt rather more tense and hard than during the early months.

Labour commenced on October 26 with infrequent pains accompanied by a show, but the pains did not become severe till the evening of October 28, and full dilatation was only reached at 9 p.m. on October 29. Throughout the labour the pain was more severe over the fibroid, which felt harder and tenser than when I had examined it a fortnight before. The second stage was short and towards the end precipitate, and I was unable to prevent a rupture of the perineum to within $\frac{3}{4}$ in. of the anus. The child, a boy, was born at 11.45, with a very long cord three times round its neck. There was rather sharp post-partum hæmorrhage, and, introducing my hand, I found placenta and membranes adherent, and had to strip them off. In doing so I felt a very small intramural fibroid in the anterior wall, and a somewhat larger submucous one near the right cornu. The perineum was stitched up, and the uterus well douched with perchloride. The pulse after labour was 86.

The following day, being obliged to go into the country, I was unable to see her until the afternoon, when she was feeling well but had a pulse of 92. On October 31, in the morning, she was still feeling well and taking her food well, and already had some milk. The pulse, however, was 98 and the temperature $100\cdot2^{\circ}$ F. The tumour was tender and painful; the lochia were scanty and serosanguineous. In the evening she did not feel so well: temperature $100\cdot4^{\circ}$ F., pulse 106; other symptoms unchanged. I ordered a dose of castor oil.

On the morning of November 1 (third day) the oil had acted well, but the patient was looking ill and anxious. The region of the tumour was very tender, so that she could not bear the weight of the bed-clothes on it; the pain in it had kept her awake the greater part of the night. The whole abdomen was somewhat tender, but only, I think, from indirect pressure on the tumour; temperature $100\cdot2^{\circ}$ F., pulse 126. Lochia almost absent, a little clear serum only. I gave an intra-uterine douche, when I found that there was a rather extensive superficial slough in the lower part of the vaginal wall on the left side. This I swabbed with pure carbolic acid. In view of the steady and progressive rise in the pulse-rate, combined with the increasing pain and tenderness over the tumour, I concluded that the fibroid was sloughing, and advised the relations that the patient's only hope of recovery, and that a very slender one, lay in immediate operation.

She was accordingly removed to the Isle of Wight County Hospital, where I operated at 3.15—sixty-three hours after the birth of the child.

Before being placed on the table she had an enema of coffee and brandy and some strychnine hypodermically; her pulse was then 132. I removed the uterus by Doyen's method. Some delay and rather free hæmorrhage occurred during the division of the vagina, owing to the thin, soft and spatulous cervix giving an unsatisfactory hold, and from the difficulty of identifying the position of the posterior fornix, which led me to cut into the lower end of the cervix. Three or four pints of saline solution were introduced into the abdomen while the wound was being closed. This caused the pulse, which had become imperceptible, to revive, and at the end of the operation, which lasted nearly three-quarters of an hour, it was not much worse than when I began. An hour later 3 pints of saline were introduced into the median basilic vein with 1 dr. of adrenalin, 1 in 1,000 to each pint. At 9.30 the pulse was 120 and feeble, but the general condition was fair.

The convalescence was without incident. The pulse for five days did not fall below 120, and was at times as frequent as 140. The gauze plug was removed on the second day, and the vaginal slough daily powdered with iodoform. The left edge of the abdominal wound sloughed a little, owing to interference with its blood supply by the scar of the former operation.

DESCRIPTION OF PARTS REMOVED.

I must apologize for the condition of the specimen, as it was placed by mistake in 10 per cent. formalin instead of 2 per cent., as I ordered. This specimen consists of the puerperal uterus, with a tumour about the size of the head of a three months' baby, slightly ovoid in shape, which has grown from the left angle of the uterus in front of the broad ligament. The round ligament runs over the back of the base of the tumour.

On slitting up the uterus its cavity is found to run up into a pocket 2 in. deep on the inner side and to the back of the tumour. There is a small submucous fibroid the size of a shelled walnut near the right cornu on the anterior wall. A second, still smaller, interstitial fibroid is situated about the middle of the anterior wall. The lining membrane is shreddy, but not unhealthy looking. The placental site is on the anterior surface, extending to the fundus, but not including the pocket

On incising the tumour there was a gush of excessively foul and stinking pus. The tumour is almost entirely converted into an irregular cavity, lined and trabeculated by yellowish-grey opaque slough. This is bounded by a layer of apparently normal fibroid, which, on the side nearest to the uterus, is $\frac{3}{4}$ in. thick, while at one point of the incision it is less than $\frac{1}{10}$ in., so that the slough is covered by very little more than the peritoneum.

REMARKS.

This case seems to me one of considerable interest, as an example of one of the accidents which may befall an apparently harmless specimen of a fibroid tumour during pregnancy. I have no doubt whatever that when I opened the abdomen in the third month of pregnancy the fibroid was in a normal condition. On the other hand, a mere glance at the condition of the specimen is sufficient to satisfy anyone that the necrotic change must have gone on a considerable time before labour.

The patient began to feel pain in the tumour on exertion about the seventh month of pregnancy, and I imagine that the central necrosis of the fibroid took place about that time or shortly before. It is interesting, too, that there should have been no symptoms suggesting any absorption of poison from the tumour till after labour, and I suppose that the unfavourable symptoms, which commenced immediately after that event, were due to the negative pressure resulting from the emptying of the uterus. This allowed the absorption from the tumour of poisons which had been, so to speak, bottled up in it by the tension of the pregnant uterus. A further point of interest lies in the ætiology of the changes in the tumour. I have not had the opportunity of making a thorough search in the literature, but I have found so little allusion to necrosis and suppuration of subperitoneal fibroids that I assume it to be a rather rare event. Pregnancy in conjunction with such tumours is comparatively frequent, and does not usually have any untoward effect upon their nutrition. Similar changes in submucous fibroids are usually ascribed to infection; and I should like to hear the opinion of Fellows whose experience is larger than my own, whether the necrosis could have been in any way brought about by the first laparotomy.

The operation was a simple exploratory one. The tumour was examined *in situ* and not brought outside the abdomen. It was

exposed to no unnecessary handling. There was no rise of temperature after operation, and the wound healed absolutely by first intention. Can it be that the ordinary handling of the tumour may so far have damaged the peritoneal surface as to allow infection to occur from the neighbouring bowels? If this were the case, the complete absence of any symptoms for four months after infection seems extraordinary.

With regard to the treatment pursued, the only alternative would have been to do a myomectomy or hysterectomy at the first operation. The former would certainly have terminated the pregnancy, and, considering the usually normal course of pregnancy, labour, and puerperium in such cases, would, I think, have constituted the more serious danger. A fortiori, I believe, that hysterectomy would have been entirely unjustifiable. After labour the symptoms did not leave any alternative.

DISCUSSION.

Dr. LEWERS said that where acute symptoms arose after labour, complicated by the presence of uterine fibroids, it was only in exceptional cases that hysterectomy was required. He quite agreed that Dr. Turner's case was one of those exceptional cases. He referred to the case he had himself recorded at the March meeting of the Obstetrical Section.¹ In that case, one of labour at the eighth month complicated by fibroids, the patient had a rigor and high temperature a few hours before labour, and the temperature remained more or less high for seven and a half weeks after delivery. Then a fibroid the size of the fist was expelled from the uterus with 15 oz. of extremely fœtid pus. Convalescence was rapidly established, and the patient made a good recovery. This patient was extremely anxious to have a living child, and unfortunately the child on the occasion referred to was stillborn. She was now pregnant again, and about to be readmitted into the London Hospital for her confinement. There were still other fibroids in the uterine wall. When she was acutely ill after the last confinement he had several times considered the question of hysterectomy, but had decided to defer it as long as possible, and the event had justified the expectant treatment adopted.

Mrs. BOYD thought that cases reported to the Section on previous occasions did not bear out the view that abortion would certainly have followed myomectomy.

Dr. HERBERT SPENCER congratulated the author on his successful treatment of the case, on his decision not to remove the tumour during pregnancy, and on his promptness in removing it when it became infected after delivery. To have removed the tumour during pregnancy would have entailed great risk

¹ *Proceedings*, 1909, ii, p. 229.

of abortion, and would have left behind a submucous tumour much more likely to cause trouble than the subperitoneal growth, suppuration in which was a very rare occurrence. It had been happily dealt with by Doyen's total hysterectomy, which presented great advantages in these puerperal cases. He thought the suppuration occurred as the result of infection in removing the adherent placenta; no doubt the tumour had previously undergone necrobiosis. There was evidence of infection in the slough in the vagina, and a section of the small submucous myoma at the fundus showed it to be deeply congested and inflamed.

Dr. TATE referred to a case of suppuration in a uterine fibroid which he operated on in October, 1906. Seven months previous to the operation the patient had a stillborn child at the eighth month. Subsequently a parametric abscess formed and burst into the vagina. The patient continued to have a hectic temperature and steadily lost flesh. The fibroid tumour, which was of small size at the time of the confinement, steadily increased in size and was very tender. At the time of the operation the tumour was as large as a seven months' pregnancy. Abdominal hysterectomy was performed, and on incising the tumour after removal it contained $3\frac{1}{2}$ pints of stinking pus with a large sloughing mass of fibroid in the centre. In this case the suppuration was clearly due to infection at the time of delivery.

The PRESIDENT (Dr. Macnaughton-Jones) said there were two important questions raised in this case: First, was the course pursued in the early period of the pregnancy the right one, and justified by the exploration of the tumour? Secondly, was the total hysterectomy called for under the conditions present after the labour? On both these points he was quite in accord with the treatment pursued. The ample discussion which they had had during the last session in the Section showed that the plan of non-interference in similar growths during pregnancy was by far the wisest and safest for the woman and child. He also felt that the subsequent radical step taken, in the face of the infective signs and symptoms, was the only course open to Dr. Turner. As to the examination of the tumour for the presence of pus, the stench disclosed at the time was to him (the President) sufficient evidence of the septic and infective nature of the tumour.

Dr. TURNER, in reply, said that he did not think that there was any possibility of the patient's recovery by natural means. In reply to Dr. Routh, he agreed that infection at the time of labour might account for the acute symptoms, but that the necrosis and breaking down of the slough must have occurred during pregnancy, as such extensive and advanced changes could not have occurred within sixty-three hours. In reply to Dr. Stevens' doubt as to the fact of suppuration, he could only say that the fluid was dirty yellow in colour and had an excessively foul odour. In reply to Mrs. Boyd, he said that his statement that myomectomy would inevitably have produced abortion was perhaps too absolute, but that the risk was certainly greater than he would have been justified in undertaking.

Note on a Case of Cystic Tumour of the Right Broad Ligament, springing from the Uterus, and apparently developed from Gartner's Duct.

By ARTHUR H. N. LEWERS, M.D.

H. M., AGED 47, was admitted into the London Hospital under my care on March 24, 1909. She had been married eight years, and had had one child, aged 7. Menstruation had occurred every three weeks, the period lasting eight days and being unaccompanied by pain.

Present illness: On admission she complained that for the last month there had been frequency of micturition during the day, and that she had been unable to empty the bladder properly in the morning. For about the same time she had also noticed a swelling in the lower abdomen, and had had pain and flatulent distension after meals.

Physical signs: On examining the abdomen, a fluctuating tumour was felt rising from the pelvis to a height of 1 in. above the umbilicus. It was fairly central in position. On vaginal examination, the vaginal portion of the cervix could only be reached with difficulty. It lay high up, and to the left. A tense swelling, which seemed to be continuous with the tumour in the abdomen, was felt bulging down the vaginal roof and right lateral fornix.

Operation (March 26, 1909): The abdomen was opened in the usual way, and the cyst, covered by the anterior layer of the right broad ligament, came into view. It was punctured, and about 2 pints of thick, chocolate-coloured, odourless fluid escaped. The relations of the cyst were carefully examined. The partially collapsed cyst was found to lie in the right broad ligament. It partly overlapped the anterior surface of the uterus, extending in that direction nearly to the middle line. The right tube and ovary were normal and lay behind the cyst, the posterior peritoneal layer of the broad ligament intervening. The bladder was drawn up, and lay in close relation to part of the anterior surface of the cyst. In order to facilitate enucleation, the collapsed cyst was tightly packed with gauze so as to define its exact limits. Enucleation was easy in every direction till the right side of the uterus about the level of the internal os was reached. Here the cyst was firmly attached to the uterus by a pedicle of uterine tissue about 1 in.

in diameter. This was divided with scissors, and during the process several small loculi the size of a pea were opened. The contents of these looked like thick yellow pus. Closer examination showed the material to be inspissated mucus, the cavities in question being lined by a secreting surface. Several large vessels were cut across when dividing the pedicle, and these had to be controlled by undersewing with silk ligatures. The deeper part of the cyst extended much more deeply downwards than is the case with ordinary broad-ligament cysts, and it was in close relation with the vaginal wall on the right side, below the level of the vaginal portion of the cervix, for a distance of at least 1 in. Nothing was seen of the right ureter during the enucleation, though it was of course looked for. After all the bleeding points had been secured, the edges of the right broad ligament were brought together with sutures, but not so as to make the cavity, from which the cyst had been removed, watertight. The left uterine appendages showed signs of chronic inflammation and were removed. The uterus was retroverted, but, apart from what has been already described as to its relation with the cyst, was otherwise normal.

The patient made an uninterrupted recovery, and went to a convalescent home on April 21.

Dr. R. Drummond Maxwell, Obstetric Registrar at the London Hospital, examined the specimen, and has given me the following report:—

MACROSCOPIC DESCRIPTION OF THE CYST.

The cyst, collapsed and shrunken after immersion in 5 per cent. formalin, measures 6 in. by 4 in., but in the recent state was the size of an adult head. A large opening that would admit a child's fist is seen at one pole, and represents the site of puncture by the trochar, the cyst wall tearing during the process. The cyst wall at the edges of this opening is thicker than elsewhere, fully $\frac{1}{4}$ in. thick, and the edges have a firm, fibro-muscular appearance. On the outer surface of the cyst are seen two smooth bosses the size of marbles. These are two small cystic cavities not communicating with the main cyst. One of them has been laid open, and a section taken through its wall; the other remains intact. At the opposite pole to these projections is seen a flat, depressed, irregular area, equal to that of half-a-crown, which represents the area where the cyst was firmly connected to the right lateral wall of the uterus. In partly enucleating and partly snipping across

the plane of the pedicle the cavity of the main cyst has been all but exposed, a very thin layer of tissue only covering this area. The lining membrane of the main cyst is rough, irregular, and brownish yellow, and in the fresh state showed signs of fairly recent hæmorrhage into its substance.

Sections for the microscope have been taken : (1) Through the wall of the small cyst projecting on the outer surface. (2) Through the thick edge of the opening into the main cavity, including the inner lining membrane of the cyst. (3) Through the sunken, crater-like area where the cyst was attached to the uterus. In this section are exposed macroscopically two small cysts filled with coagulated contents.

Report on the Sections.

Section (1) through the projecting cyst on the outer surface : The cyst has mucoid contents, and is lined by a single layer of columnar (? ciliated) epithelium. Probably the appearance of cilia is due to inspissated mucoid secretion adherent to the free border of the cells. The stroma of the cyst wall consists chiefly of bundles of cells with short oval nuclei, staining yellow with Van Gieson's stain, with fine fibrils between the nuclei. The nuclei are not so close as in ovarian stroma, but do not seem to be as long as those of involuntary muscle.

Section (2) : The sections through the thick wall of the cyst, stained with Van Gieson's stain, show it to be composed of involuntary muscle fibre and connective tissue, as in the deeper layers of the uterine wall, but arranged in a more laminated form. The inner aspect or lining of the main cyst has no cell lining ; it is partly smooth, partly ragged, and is not necrotic. It consists of connective-tissue fibrils, separating and lying between which are red cells and many fine lymphocytes with granular and thready débris.

Section (3) through the hilum : Muscle bands are here rather more definite than in Section (2). The lining membrane of the large cyst is smoother, but has no definite cell lining. In the stalk are some very large vessels and some tubules lined by columnar epithelium. Some of these tubules have wavy, crenated outlines. Others are dilated to form cysts with coagulated contents. The cyst has a fibro-muscular wall of structure identical with that of the uterus, and may be considered as growing in its wall. The lining membrane of the large main cyst, though not necrotic, has lost its cellular lining, probably owing to pressure. The numerous secondary cysts in the wall all show the

same high columnar epithelial lining secreting a mucoid albuminous substance, in places coagulated, and forming retention cysts.

The sections support the view that the tumour represents a collection of dilated ducts and cysts in the uterine wall, probably arising from the intra-uterine portion of the Gartnerian duct.

DISCUSSION.

Mr. ALBAN DORAN referred to Unterberger's remarkable instance of a bilobed cyst of Gartner's duct published in the *Monatsschrift für Geburtshilfe* last May.¹ There were apparently two cysts, and on pressure on the left tumour sanious watery fluid issued from the os externum. A vaginal operation proved a failure; a fortnight later Unterberger opened the abdomen. The tumour was so intimately connected with the supravaginal part of the cervix that the uterus was removed as well. The tumour consisted of two cystic bodies apparently of bilateral development. On dissection, however, it was found that the right cyst had a well-formed duct which ran through the wall of the cervix opening into its canal; whilst the left cyst also had a duct, but it opened into the right duct, passing behind the cervix. Hence the tumour must be classified as a bilobed cyst of the right duct of Gartner. In this case the ducts were saved, but unfortunately the patient was lost. In Dr. Lewers' case the patient recovered, but the cyst had been dissected off the uterus and the ducts were therefore cut through. But in Unterberger's case there was long-standing infection before operation, the contents of the cysts being fætid; and in that exhibited this evening, though for surgical reasons it had been found necessary to divide the attachments of the cyst, there could be little doubt that it was homologous to Unterberger's tumour.

Dr. AMAND ROUTH alluded to five cases of cysts developed from Gartner's ducts which he had described in a paper read before the Obstetrical Society in 1894.² His own case and those of Watts and Veit were cases of communicating vaginal and broad-ligament cysts. In another (Milton's) Gartner's duct opened on to the antero-lateral wall of the vagina; and in Lawson Tait's case, both Gartner's ducts opened out in the vestibule on either side of the urethral orifice. In the latter cases probes could be passed into the broad-ligament regions. He knew of no means of deciding whether a parovarian cyst was developed from Gartner's duct or from one of the vertical tubules of the parovarium, except from the way that the former tended to burrow also along the antero-lateral wall of the vagina. He asked for more evidence that Gartner's duct habitually entered the uterine substance.

Dr. DRUMMOND MAXWELL said that, as registrar to the hospital, he had had the opportunity of examining the tumour, which at first sight had appeared to be an intraligamentous fibroid undergoing cystic degeneration. It was

¹ 1909, xxix, p. 587.

² *Trans. Obstet. Soc. Lond.* (1894), 1895, xxxvi, p. 152.

only when sections were taken from its walls that its interesting origin was suspected, and he thought the evidence of the accompanying microscopic sections warranted the view that the tumour was of developmental origin, and arose in the intra-uterine portion of Gartner's duct. The evidence rested on several considerations. In the first place, there was no doubt that the tumour was enucleated from the uterine wall by the operator's scissors, and the sections of the wall of the main cyst showed a tissue stained by "Van Gieson" identical with uterine wall. Unfortunately the main cyst had no epithelial lining. In the light of other sections, in all probability it had originally possessed one, but this had been destroyed by hæmorrhagic and pressure changes and was too necrotic to prove the point. The main evidence of Gartnerian origin rested on the lining membrane of two small cysts projecting from the outer wall of the main cyst. These two cysts contained a clear albuminous secretion, and had a lining membrane of high columnar epithelium on which cilia could be seen in places. Sections also were shown on the table from that part of the tumour which had been severed from the lateral wall of the uterus during enucleation. These latter sections had passed through several macroscopic cysts averaging about $\frac{1}{4}$ in. in diameter. There were also seen scattered through the field gland lumina—some circular, others with wavy crenated outlines lined by delicate high columnar epithelium ciliated in places. The cysts contained coagulated secretion identical with that of the previously mentioned cysts. One could exclude the oophoron and epoophoron from playing any part in the pathology of this tumour, since the ovary lay remote from the tumour, and was not encroached on either during or after the operation. The tumour therefore seemed to be one of Recklinghausen's type, in which the main bulk, instead of being directly incorporated in the uterine wall, had projected out laterally into the cellular tissue of the broad ligament.

Mrs. BOYD was interested in the observation that the ureter was not seen. In a similar case of her own, where a large cyst presented in the abdomen and burrowed deeply into the recto-vaginal septum down to the pelvic floor, wrapping the uterus round so that it appeared both in the anterior and posterior vaginal vault, the ureter was stretched out over the outer abdominal surface of the cyst.

Dr. LEWERS thanked Mr. Doran for giving him the reference to Unterberger's case. It seemed to be a similar one to that recorded in his own paper. Unfortunately Unterberger's case had ended fatally, and this seemed probably to have been due to his attacking the tumour from below first ineffectually, and later on having to deal with the tumour from above. Dr. Lewers thought it best to remove tumours of the kind by the abdominal route, as he had done in his own case. With reference to Dr. Routh's remarks, Dr. Lewers did not think he had quite appreciated the facts that the wall of the cyst was formed of uterine tissue, that it was connected by a stout pedicle of uterine tissue to the uterus at the level of the internal os, and that the small secondary cysts in the wall of the main cyst were lined by columnar epithelium.

Specimen from a Case of Carcinoma of the Ovary.

By L. GARRETT ANDERSON, M.D.

THE specimen consisted of uterus and appendages removed after death from a single woman aged 34. The uterus and the left tube and ovary appeared normal, and microscopic sections prepared from them corroborated this. The right ovary was converted into a solid tumour weighing $7\frac{3}{4}$ lb. This appeared to be a primary carcinoma of the ovary, which is a somewhat rare condition. The patient had noticed that her abdomen had increased in size for a year, and latterly she had suffered on various occasions from severe attacks of abdominal pain. An exploratory laparotomy was performed, but when the growth was found to be malignant it was thought inadvisable to proceed, as the operation would have been serious and the condition was obviously hopeless.

The exhibitor remarked on the entire absence of free peritoneal fluid. She thought that the absence of ascites could in no way be regarded as an important diagnostic point. Many large malignant ovarian tumours, although of very rapid growth and extreme malignancy, did not cause ascites, whereas non-malignant papillomata were usually associated with a large amount of effusion. She quoted figures from the records of the New Hospital for Women.

The PRESIDENT said that Dr. Herbert Spencer had referred at the previous meeting to a specimen of his on which a committee of the Obstetrical Society had reported, deciding that the tumour was not carcinomatous. Though it was removed several years back, he had since then learned that the patient was in excellent health, thus confirming the view taken at the time. He had brought for inspection two adeno-carcinomatous ovaries, with subperitoneal myomatous tumours, removed from the same patient—the case he had referred to at the last meeting. He had just had a patient with extensive adeno-carcinoma and a large myoma of the uterus which was inoperable. He had made an exploratory incision after first curetting. The portions of the uterus curetted were reported to be benign, but the metastatic growths which he removed on exploration were taken from secondary adeno-carcinomatous masses transplanted from the ovaries. In this case there was only a very small quantity of ascitic fluid, as emphasized by Dr. Garrett Anderson. An important practical point was the frequency with which both ovaries were affected, and the necessity for caution before determining not to remove the apparently sound ovary.

Tumour of the Uterus, of Doubtful Nature.

By J. BARRIS, F.R.C.S.

THE specimen, for which I am indebted to Dr. Griffith, is that of half a uterus, showing a growth in the anterior lip of the cervix. The tumour is of a dead white colour, oval in shape, measuring $1\frac{1}{2}$ in. in its greatest diameter. It is not clearly marked off from the surrounding structures.

This specimen was removed by a modified Wertheim's operation from a woman aged 51, the mother of six children. The menopause occurred at the age of 45. Six years later she commenced to suffer from irregular uterine hæmorrhage, bleeding on coitus, vaginal discharge, and pain in both iliac fossæ. The clinical history is, then, that of a carcinoma, but microscopical examination renders this doubtful and suggests the possibility of the tumour being a sarcoma. In favour of this supposition there are the following points: The growth does not appear to originate from the epithelium; the shape of the cells is more like sarcoma than carcinoma; the relation of cell substance to nucleus suggests sarcoma, in that the nucleus mainly fills the cell; a few blood-spaces can be seen, surrounded by thin walls; at the time of operation no enlarged glands were detected.

Report of Pathology Committee.—We are of opinion that the growth is a spheroidal-celled carcinoma of the cervix, of a type uncommon in this situation.

Myxofibroma of an Ovary removed under Spinal Anæsthesia.

By WALTER TATE, M.D.

THE specimen shown was removed from an unmarried patient aged 49. The patient's mother died of cancer. The patient had pleurisy in the right side at Christmas, 1906. In February, 1908, she had pleurisy with effusion in both sides, following influenza. The right side was tapped four times and the left side three times, 22 pints of fluid in all being removed. At this time a solid tumour, which was

thought to be a uterine fibromyoma, was first discovered. The periods remained quite regular till June, 1909, when they began to be scanty, and from this time the abdomen began to increase steadily in size. Owing to the large size of the abdomen and the interference with breathing the patient was recommended for hospital treatment, and was admitted to the medical ward of St. Thomas's Hospital on October 11, 1909.

On admission the patient looked very ill, her breathing was much distressed, and she had to sit up in bed on this account. Her abdomen was very distended owing to the large amount of ascitic fluid present, and there was also evidence of fluid in both pleural cavities. On October 13 the abdomen was tapped and 12 pints of ascitic fluid removed. After removal of the fluid a smooth, hard and very mobile tumour rising out of the pelvis could be palpated in the lower part of the abdomen. On October 15, 3 pints of clear straw-coloured fluid were removed from the right pleural cavity, and on October 20, 2½ pints were removed from the left pleural cavity. The fluid was examined by Dr. Dudgeon, but the cell contents were too degenerate to admit of any diagnosis. There was some question as to whether the condition in the lungs was due to a new growth secondary to a malignant growth in the abdomen. When I examined the patient on October 20 the uterus was small and atrophic. The lower pole of the movable abdominal tumour was found to depress the vaginal fornix. The tumour, however, was quite separate from the uterus and was thought to be a solid tumour of the ovary, either a fibroma or a sarcoma. Abdominal exploration was recommended, but, owing to the patient having so recently had extensive effusion into both pleuræ, it was not thought safe to give a general anæsthetic.

On October 21, 1909, 1 c.cm. of 10 per cent. stovaine was injected between the second and third lumbar vertebræ. Within a minute and a half the patient was anæsthetic over the operation area, and after cleansing the abdominal wall an incision 7 in. long was made above the pubes. On opening the peritoneum a quantity of ascitic fluid escaped. The hard tumour, which involved the left ovary, was removed after the pedicle had been clamped. The right ovary contained a small cyst, and was removed, as the nature of the tumour of the right ovary was doubtful. After cleansing the pelvis the wound was rapidly closed in layers. The operation lasted twenty minutes. The patient experienced no discomfort during the operation. Sensation and power of movement returned in the legs within half an hour after the operation. She

complained of smarting pain in the wound, but was otherwise comfortable.

Recovery was quite uneventful, and on examination of the patient four weeks later, before leaving the hospital, the abdomen was quite free from ascites and there was no return of fluid in the chest.

The tumour removed measured 8 in. by 8 in. by $4\frac{1}{2}$ in. ; its weight was 4 lb. 7 oz. ; it was elliptical in shape, its surface white and glistening. Some large superficial veins could be seen on the surface. On section the lower part was found to consist of a solid mass of uniform consistence. The upper part was made up of several hard nodular masses alternating with softer portions and some cavities containing clear fluid. Two of these cavities measured 2 in. by $1\frac{3}{4}$ in. by $\frac{3}{4}$ in. and 2 in. by 1 in. by 1 in. respectively. They appeared to have a lining membrane. The microscopic examination of the tumour by Dr. Dudgeon proved it to be a myxofibroma.

Although when admitted to the medical ward the clinical aspect of the case suggested the diagnosis of malignant abdominal tumour with secondary growth in the lungs, it is evident that the attacks of pleurisy, dating back three years, were quite independent of the abdominal tumour, inasmuch as the latter had only caused symptoms for three or four months. It is, however, quite easy to understand how the large amount of ascitic fluid associated with the fibroma of the ovary lighted up an old-standing pleurisy and caused the effusion into both pleural cavities. The fact that there was no sign of any return of the effusion into the pleural cavity after the removal of the tumour confirms this view.

Primary Carcinoma of the Fallopian Tube associated with Acute Inflammatory Mischief.

By WALTER TATE, M.D.

THE patient, Mrs. C., aged 52, had had three children, the last seventeen years ago. The menopause occurred at the age of 48, and for the past two years there had been a yellowish vaginal discharge, occasionally blood-stained. There had also been some pain and discomfort in the pelvis during the same period. The patient, however, kept in good health till the commencement of her present illness, which occurred within twenty-four hours of a motor accident, in which she

received a severe shaking. On the day following the accident she did not feel at all well, and complained of some abdominal pain. She was seen by Dr. White, of Putney, who found some tenderness over the lower abdomen and a temperature of 100° F. On the following day the patient had a severe attack of pain in the lower part of the abdomen on the left side, accompanied by vomiting and serious collapse. I saw her in consultation at 3.30 p.m. on the same day. There was some distension of the lower abdomen and marked tenderness over the left iliac and hypogastric regions. The uterus was retroverted and its mobility impaired. A tense swelling as large as a hen's egg was felt in the left posterior quarter of the pelvis, and a similar swelling in the situation of the right appendages. Palliative measures were tried for a time, but, as the patient had much abdominal pain during the night, it was decided to explore the abdomen on the following morning.

Abdominal section was performed on July 21, 1907. After opening the abdomen, there was a good deal of distension of the coils of intestine in the lower part of the abdomen, and after separating some adhesions of omentum and bowel on the left side of the pelvis, several ounces of turbid serum escaped from round the left appendages. The left Fallopian tube was distended and appeared to contain pus. After transfixion of the broad ligament, the tube and a portion of the ovary adherent to it were removed. After the separation of some adhesions round the right appendages, the tube and ovary of the right side were also removed.

Owing to the general condition of the patient and the acute inflammatory conditions present it was not thought wise to attempt removal of the uterus, which was only slightly enlarged. Moreover, at this stage of the operation, the malignant nature of the tubal disease was not suspected. After the cleansing of the cavity of the pelvis and adjoining coils of intestine with warm saline solution, the wound was closed in layers without drainage.

The parts removed included the right Fallopian tube which was 5 in. long and somewhat tortuous. It measured 1 in. in diameter. Its outer surface was injected and presented a few torn adhesions on the surface. On section the wall was found considerably thickened and the cavity of the tube filled with a very soft friable growth, which broke away from the wall when touched. The left tube, which was smaller than the right, presented a similar appearance externally. On section, it was found to contain about 1½ oz. of muco-purulent fluid. The inner surface of the tube was covered with lymph. A few warty

growths were seen springing from the inner surface of the tube. These were very friable and resembled papillomatous growth.

The microscopic report by Dr. Cuthbert Lockyer states: The larger right tube is the seat of a glandular carcinoma of the columnar type. There is extensive development of fibrous tissue, and the cancer cells group themselves into masses and hollow tubes in alveolar spaces within this fibrous stroma. The smaller tube (left) is thickened and infiltrated (by inflammatory products) in all its coats. The mucous membrane is almost totally destroyed and replaced by granulation tissue. There is no sign of malignancy in this tube.

The uterine discharge entirely ceased after the operation, and the patient made an uneventful recovery. She was last seen by the writer on October 5, 1909—that is, two years and three months after the operation—and was then perfectly well and had put on weight since her operation.

Report of Pathology Committee.—We are of opinion that the specimen is a papillary, columnar-celled carcinoma of the Fallopian tube. There is every reason to regard the growth as primary.

DISCUSSION.

Mr. ALBAN DORAN stated that he had operated on a patient subject to primary cancer of the Fallopian tube in 1899. The Section must admit that he had not shown undue haste in making the case public, but he intended to report it in a communication which would shortly appear in the *Journal of Obstetrics and Gynæcology*, together with tables which would bring the number of reported cases up to 100. He turned the attention of the Section to Boxer's "Beitrag zur Kenntnis des Tubenkarzinoms" in the November number of the *Monatsschrift für Geburtshülfe*.¹ He showed how, in two cases of cancerous tubo-ovarian cysts where the new growth had without doubt originated in the tube, the growth had extended to the ovarian portion by implantation. The Fallopian tubes had become thickened and dilated owing to chronic inflammation. In a third the canal of the right tube had acquired a communication with a cancerous ovarian cyst and had become infected by implantation, bearing patches of psammomatous cancer. Certain tubules, lined with epithelium, had insinuated themselves into the muscular coat of the tube; they represented changes due to chronic salpingitis. Mr. Doran had figured a section of a cancerous tube, showing a tubule of this type, in his report on Mr. Knowsley Thornton's case of primary cancer, published over twenty years ago in the thirty-ninth volume of the *Transactions of the Pathological Society*, though at that time he did not clearly understand the

¹ 1909, xxx, p. 549.

significance of the tubules. Boxer found that these tubules in the right tube in his specimen carried infection into the spaces of the connective tissue of the tube. He examined the left Fallopian tube and found precisely similar psammomatous cancerous growths growing freely from its inner wall, with the characteristic tubules, infected and communicating with connective-tissue spaces. There were no cancerous growths in the endometrium, or in the muscular or serous coat of the uterus, or on the pelvic peritoneum. Therefore Boxer concluded that the left tube had become infected through the lymphatics and that it was now easy to understand why cancer of the tube was so often bilateral. In Dr. Walter Tate's case there had been previous inflammation of the tubes. Hence that case and Boxer's important researches supported the theory advanced by himself in 1888, and maintained by Snger six years later, that salpingitis greatly favoured, and seemed to be a usual, if indirect, cause of, cancer of the Fallopian tube.

Dr. HERBERT SPENCER hoped that the specimen and microscopic sections would be submitted to the Pathology Committee. In view of the absence of recurrence it was important to be quite sure that the growth was carcinoma. His own experience had been much less fortunate. He had operated on three cases of primary carcinoma of the Fallopian tube. The first patient, aged 64, who had a small secondary nodule in the vagina, had succumbed after Wertheim's operation. No other growths were found at the post-mortem examination. In the second case the cancerous tube complicated a uterine myoma as big as a cocoanut. Removal of the tube was followed by recovery, but the patient died just a year after the operation with secondary growths in the peritoneum and abdominal scar. In the third case the growth had extended to the left broad ligament, which was removed. In this case also recurrence occurred within a few months. He showed drawings of the last two specimens. He purposed publishing the three cases in the *Journal of Obstetrics and Gyncology of the British Empire*. He might add that in addition to these three cases he had had another, in which both Fallopian tubes of a myomatous uterus were filled with a whitish growth which to the naked eye resembled cancer and which contained tubular structures lined with columnar epithelium. The patient was quite well two years after their removal, but he felt sure that the "growth" was the result of chronic inflammation and was not malignant.

The PRESIDENT said that he had met with only one case of primary carcinoma of the tube. It was unilateral. A full report on the diseased tube had been made by Dr. Cuthbert Lockyer. A drawing of the condition and his report were put on record. The growth was enclosed in a thin capsule, which was continuous with the wall of the tube, which at its outer end was intact and then thinned out to form a capsule of a dense, carcinomatous growth. The first recorded case was that of Orthmann in 1888. This was associated with abscess of the ovary. From the statistics collected by Mr. Doran it would therefore appear that in the twenty-one years there had been altogether one hundred cases recorded, which showed that the condition was comparatively a rare one.

Quadruplets born at the Twenty-sixth Week.

By CLIFFORD WHITE, M.D.

THE mother, a healthy woman, aged 28, had had seven children and three miscarriages. The last child was only ten months old when these children were born, and, as there was the amenorrhœa of lactation, she was unable to exactly say how far pregnant she was, but thought herself to be twenty-six weeks. She quickened eight weeks before delivery. The early stage of the labour, which came on on October 5, 1909, was complicated by hydramnios; but after the membranes had ruptured, the first child, a stillborn male, was delivered within three hours of the first onset of pains. There was no excess of liquor after the first bag of membranes had ruptured. The second child, also a stillborn male, was born twenty minutes after the first. The third, a female, was delivered twenty minutes later; it lived five and a half hours. There was a delay of an hour and a quarter before the fourth was born, and Dr. Osborne had to rupture the membranes, after which it was rapidly delivered. It was a male, and lived half an hour. All the males were vertex presentations; the female was a breech.

The placentas show that three ova are represented, the first two small children being uniovular twins. The twin placenta and that of the third child came away together; that of the fourth child came away fifteen minutes later. There was no post-partum hæmorrhage. The puerperium was normal. As regards the family history, there have been several twin births on the husband's side: an aunt had twins on two occasions. On the patient's side, another aunt and a sister each had twins once. The lengths and weights of the fœtus are:—

(1) Nine inches long	Weight 10½ ounces
(2) Ten inches long	„ 11 „
(3) Fourteen inches long	„ 18 „
(4) Thirteen inches long	„ 18 „

I have to thank Dr. T. G. Stevens, who was in charge of the Queen Charlotte's out-patients at the time, for permission to show this specimen this evening.

Placenta and Membranes of Quadruplets.

By J. ABERNETHY WILLETT, M.D.

THIS specimen was obtained from a patient who had been married fifteen years and had had ten children and one miscarriage previously (all single pregnancies). The last period ended on January 16, and on April 30 she miscarried. The first three foetuses were born in rapid succession, and the fourth with the placenta about an hour later. Until the placenta came away there was a good deal of hæmorrhage. With the exception of a niece (eldest sister's daughter) there is no instance of a multiple pregnancy either in her own or her husband's family. In the specimen the chorion appears to be single, though one extremity of it is but loosely attached to the main mass. On its outer surface there is a small area in which the chorionic villi are more distinct. On its inner surface there are four foetuses, each lying in its own amniotic sac. Three of the embryos are well developed and are apparently of about the third month. They vary slightly in size, the largest being attached to the semi-isolated portion of the chorion. The fourth embryo is shrivelled and headless, and seems to have been dead for some little time.

Dr. R. H. HODGSON drew attention to the great difference in length of the foetuses, one being no less than five inches longer than another, and no two being alike. If these quadruplets were the result of one conception, then such difference rather shook one's belief in the generally accepted theory that the length of a foetus may be taken as the guide to the length of the pregnancy.

Bilateral Tuberculous Pyosalpinx.

By HERBERT J. PATERSON, F.R.C.S.

A WOMAN, aged 23, suffering from what was supposed to be a third attack of appendicitis, was admitted into the London Temperance Hospital under my care in September, 1907. The catamenia had been regular, but scanty and painful. She had been married fourteen months, but had not been pregnant. At the age of 10 she was in the Middlesex Hospital with peritonitis. In March, 1907, she was in another hospital with an attack of pain in the right iliac fossa, accom-

panied by vomiting. On September 2, 1907, she was seized with pain in the right iliac fossa accompanied by diarrhœa. At first she improved, but became worse on September 8, and was admitted into the hospital on the following day. On admission her temperature was 100·8° F., and she had marked pain, tenderness, and resistance in the right iliac fossa. She steadily improved after admission, and her temperature was normal on September 16. On making an examination a week later I found a swelling in the right posterior quarter of the pelvis about the size of a walnut, and in the left posterior quarter a tender swelling about the size of a tangerine orange. Both swellings were closely connected with the uterus. A diagnosis of tuberculous pyosalpinx was made and operation advised. On October 1 I opened the abdomen in the middle line. The intestines in the lower part of the abdomen were extensively studded with miliary tubercle, and were matted together with old adhesions, so that access to the pelvis was rendered very difficult and tedious. After separation of the adhesions a double pyosalpinx was exposed. The appendages and uterus were removed. The patient was very ill for twenty-four hours, but afterwards, thanks to skilful nursing and continuous proctoclysis, she made an uneventful recovery. Fortunately, with some assistance, she was enabled to get away to the seaside for six months, and she soon regained her health, and is at the present time (I saw her on December 8, 1909) perfectly well and strong. There can be little doubt that the first attack of peritonitis was tuberculous, and the long interval between the first and second attack is of interest. The case further illustrates how complete a recovery a patient may make from extensive tuberculosis if the primary focus be removed, and if the patient can get away to the seaside after operation.

The PRESIDENT reminded the Section that he had brought three cases of primary tuberculosis of the Fallopian tube before the Fellows during its first session. In one of the three both tubes were affected. In another, through a second operation some years after, the other *adnexa* were proved to be free of tubercle; all three patients were now alive and in excellent health. This proved the importance of early and complete operation in such cases.

Mr. PATERSON also exhibited an apparatus he had devised for continuous proctoclysis, a description of which appeared in the *Lancet*, October 9, 1909.

The PRESIDENT said that he had had perfect satisfaction with the apparatus he had shown in October. With it he had given as many as from 15 to 18

pints of saline in the twenty-four hours, and had kept a patient under proctoclysis on and off for three days. Mr. Paterson's contrivance was a more complete one, inasmuch as it showed automatically the quantity being used and the temperature. It should, however, be remembered that with a very simple apparatus the continuous method could be carried out. In fact with an ordinary rubber douche bottle, periodically replenished, and connected by a rubber tube with the rectal pipe, the tube interrupted at a part with a Leiter's regulator, which could lie in a bowl of water at a given temperature, any intelligent nurse could regulate the quantity and temperature of the saline. The important point after the quantity was the warmth of the fluid; if over-hot it tended to induce a movement of the bowel. As this treatment was often required in emergency and in the onset of a septic peritonitis, it was most important for the practitioner to be able to start it without the delay of procuring any elaborate appliance. Samuel Newman had shown that this could be achieved by means of two connected funnels, the supply being regulated by plugs. Elbrecht's apparatus,¹ which had an electric heater, was a very complete one, but again had the disadvantage of complication. Moynihan's pewter rectal pipe was superior to any rubber one. They had in proctoclysis the greatest recent addition to gynæcological and abdominal surgery. This view was that of Moynihan, who used it so largely in his gastric and bowel operations. This debt of surgery was due to J. B. Murphy.

Posterior Cæsarean Section followed by Total Hysterectomy for Fibroids.

By HERBERT R. SPENCER, M.D.

S. R., AGED 37, was admitted to University College Hospital on February 11, 1909. She was about four months pregnant, and had had a flooding at the fifth week. She complained of much pain and tenderness over the uterus, which was abnormally large, was especially developed in the right side, and reached up to a height of 6 in. above the pubes. The uterus was hard all over and very tender to the touch. A sub-peritoneal myoma, as big as a pigeon's egg, could be felt above the right Poupert's ligament. There was colostrum in the breasts, and the cervix was typical of pregnancy. The uterus was too tender and too hard to allow ballottement to be obtained. From the hardness and abnormal size of the organ it was thought that other myomata were present, though no definite nodule, except the one mentioned, could be felt.

¹ *Journ. Amer. Med. Assoc.*, Chicago, 1909, lii, p. 1248.

The patient remained in the hospital till March 17, and under rest and sedatives (bromide of potassium and, on a few occasions, morphia) the pain was somewhat relieved; but the tenderness remained, especially over the sub-peritoneal tumour. The temperature remained normal throughout. The patient was a fairly healthy woman, but had suffered from abdominal pains, on and off, since her last full-term child was born, thirteen years ago; the pain was aching in character and was felt especially on the right side. The pelvis was of fair size, the external measurements being normal; the promontory was reached with difficulty owing to the contraction of the outlet and the narrowing of the pubic arch; the diagonal conjugate was estimated at $4\frac{1}{2}$ in. Her labours had all been difficult. The first child was born by the breech; instruments were used to deliver the after-coming head; the child was born alive, but the mother nearly died. The second child was delivered with difficulty with instruments; the child was injured in the head and is delicate; there was a good deal of post-partum hæmorrhage. The third child also was delivered with great difficulty: both its arms were broken, and the head was marked at birth, and remained so. This child is very dull and silly. In 1899 the patient was admitted, pregnant, into a London hospital, having lost blood from the vagina continuously since the fifth week. She suffered great pain both before and after labour, which was induced at six and a half months. A small lump (? fibroid) was removed on the day after delivery. Menstruation had always been regular (lasting eight to ten days), was somewhat excessive in amount, and accompanied by pain.

The patient was re-admitted to University College Hospital, and on July 10, 1909, when the pregnancy was computed to have reached thirty-eight and a half weeks, Cæsarean section was performed and the uterus was removed, the ovaries and tubes being retained. A larger abdominal incision than usual was made, and through this the uterus was withdrawn and incised along the posterior wall of the lower part of the body. After the child and placenta had been extracted the incision was enlarged a little into the upper cervix: the left forefinger was then passed through the incision and hooked over the posterior lip of the cervix, the posterior fornix incised over the finger, and the uterus was removed with scissors by Doyen's method. The peritoneum was closed by a purse-string suture. The operation lasted fifty-five minutes. The wound healed by first intention, and a good recovery was made by mother and child. The infant, suckled by its mother, weighed 7 lb. $2\frac{1}{2}$ oz. at birth, and 7 lb. $14\frac{1}{2}$ oz. on August 3, when they left the

hospital. Both were quite well on November 30, and the mother had had no abdominal pain since the operation.

The uterus measured 18 cm. by 16 cm. by 10 cm., and weighed 2 lb. 14 oz.; the placenta in addition weighed 1 lb. 4 oz. In the anterior wall was seen a fibroid which on the surface measured 6 cm. in diameter; but the tumour itself, imbedded in muscle, was only 3·4 cm. in its longest diameter, was white, and to the naked eye showed no sign of degeneration. Two similar growths of the size of small marbles were found in the uterine wall on incising it. The uterine wall was very thick, the posterior wall of the body was 5 cm., the fundus 3 cm. in thickness, and the section of the tissue was streaked with white lines. The microscope showed extensive hyaline degeneration of the uterine muscle, which stained badly and was in places vacuolated. The myoma was also degenerated, but to a smaller extent than the uterine muscle.

The features of the case on account of which the specimen was shown were the large size and great thickness of the uterus, although the tumours were small, the great pain and tenderness which were present (in the absence of red degeneration), the degenerated condition of the uterine muscle, and the method of incising and removing the uterus. In ordinary conditions I regard the withdrawal of the uterus from the abdominal cavity before section as faulty technique, and I think in the case described it would have been preferable to make an anterior incision with the uterus in the abdomen, to remove the child, and then, withdrawing the uterus, to hook the finger on to the cervix through a stab-hole in the posterior wall. I am unable to offer any explanation of the degeneration of the uterine muscle. It cannot be a post-mortem change, for it has never been met with in over a hundred cases of fibroids similarly prepared, and its appearance to the naked eye is peculiar and suggests a fibrotic change, which, however, Van Gieson's stain showed not to be present.

Obstetrical and Gynæcological Section.

January 13, 1910.

Dr. H. MACNAUGHTON-JONES, President of the Section, in the Chair.

On the Proportion of Malignant to Innocent Ovarian Growths, Founded on a Series of 150 Cases.

By MARY A. SCHARLIEB, M.S.

WITH the greatest reluctance I am obeying the order of our President in offering you a short paper on the relative proportion of malignant and simple ovarian tumours, in the hope of inducing a discussion chiefly on classification and diagnosis. The number of my cases dealt with in this paper is small, only 150, but they are consecutive. I have not been able to include those of other women-surgeons, as I hoped to do had time permitted. My own cases may be classified as :—

SIMPLE.				MALIGNANT.			
(1) Paucilocular	48	(1) Papilliferous cysto-carcino-			
(2) Multilocular	42	mata	13
(3) Adenomata	10	(2) Adeno-carcinomata	7
(4) Papillomata	2	(3) Sarcomata	4
(5) Dermoid	16	Uncertain	1
(6) Corpus luteum	2				
(7) Lutein cyst	1				
(8) Fibroma	3				
(9) Fibromyoma	1				
			125				25

Note.—The malignant cases were examined macroscopically and microscopically, with the exception of Nos. 1, 2, and 4. The non-malignant cases were examined macroscopically, but not invariably microscopically until 1902. This embraces about half of the presumably non-malignant cases. The conclusion is therefore justified that *at least* one-sixth of the total number were malignant.

In this series of 150 cases I have not included any tumour or cyst smaller than a hen's egg, and only under exceptional circumstances anything smaller than a lemon, thus excluding retention cysts, both blood-cysts and dropsical Graafian follicles. I was, of course, quite aware that in a good many cases the growth was malignant. This was proved by the microscopic examination of the specimen subsequent to operation, and in a considerable number of cases it was proved in an equally convincing manner by recurrence of the growth in an unmistakably malignant form; but I must confess that I was not prepared to find that one-sixth of all my cases could be so classified. It is interesting to find that, in 16 consecutive cases reported by Dr. May Thorne, 3 were malignant and 13 simple; while in another 16 reported by Mrs. Vaughan-Sawyer, 2 were malignant and 14 simple. Taken together, there were 5 malignant in 32 cases, almost exactly the same proportion as in my own series. I am quite aware that the numbers are too small to justify any certain conclusion, but the really startling proportion in which the growth was malignant seems to me to justify the opinions that are steadily gaining ground in the profession—first, that every case shall be carefully recorded; second, that in every case the specimen shall be examined by an expert pathologist; third, that surgeons shall, in all instances, do their best to ascertain the subsequent history of their patients; and, fourth, that ovarian growths must, when possible, be delivered without tapping or incision. Of the 25 malignant cases, 6 died within a month of operation, 8 within a year, 3 within two years, 1 within three years, 1 within five years; 1 operated on in November, 1904, is still alive and in good health, and of 5 there is no history after they left the hospital apparently well. Of the 125 innocent cases, 2 died soon after the operation, 1 from the slipping of a ligature, and 1 (a suppurating cyst universally adherent and not removable) died of exhaustion.

In going over the records of the 25 malignant cases, I find that 13 were papillomata—that is, papilliferous cyst-adenomata—7 were considered by the pathologists to be adeno-carcinomata, 4 sarcomata, and of 1 the nature was uncertain. I propose to read very brief notes of these 25 cases:—

(1) Mrs. E., aged 52, no children. Suffered from papillomatous disease of both ovaries. At the time of operation the disease was found widely disseminated on the pelvic organs, the bladder especially being invaded. This patient died a few days after operation.

(2) Miss D., aged 54. Both ovaries papillomatous, but the papillomata grew inside the cysts and had not perforated their walls. The only disease external to the cysts was one small, warty growth on the parietal peritoneum. She died one year after of generalized cancer of the peritoneum.

(3) Mrs. M., aged 49, three children. She had sarcoma of both ovaries. At the time of operation all the other organs appeared to be normal, but she died of sarcoma of the uterus, which first appeared about a year after the ovariectomy.

(4) Mrs. A., aged 45, three children. Had two innocent-looking ovarian cysts; they were large and contained a quantity of clear fluid, and had adenomatous intra-cystic growths. There were no adhesions, and there was no appearance of disease anywhere in the abdomen or pelvis. About eight months later a malignant growth sprouted through the lower angle of the cicatrix, and on examination was found to fill the entire pelvis.

(5) Miss B., aged 35. Papillomatous disease of both ovaries, widely disseminated growths in the abdomen. Left the hospital well; no subsequent history.

(6) Mrs. G., aged 33, one child. Had papillomata of both ovaries. There was a quantity of ascitic fluid in the abdomen and dense adhesions. She left the hospital well; there was no subsequent history.

(7) Mrs. T. S., aged 40, two children. The patient's symptoms were chiefly those of pelvic discomfort, and although she had some indigestion it was thought to be secondary to the very obvious pelvic tumours. On opening the abdomen both ovaries were seen to be transformed into dense white, solid masses, about the shape and size of lemons; they were freely movable, there was no other evident disease, and there was no ascites. Patient made a good recovery, but died of intestinal obstruction ten months later. In the course of an operation to relieve the intestinal obstruction cancer of the stomach and transverse colon was found.

(8) Miss P., aged 64. She had malignant cysts of both ovaries; there were secondary deposits in the omentum and peritoneum. She died about two months after operation.

(9) Miss Co., aged 69. This was a somewhat interesting case. Patient was sent up to me from the country, and during her cab drive from Charing Cross felt something give way inside. At the time of the operation the abdomen was found to be full of fluid mixed with colloid material which had escaped from a large ruptured cyst of the left ovary. She bore the operation well and seemed to be going on satisfactorily, but died suddenly from syncope on the twelfth day.

(10) Miss Ch., aged 68. Papillomata of both ovaries. There was ascites, also growths in the omentum and mesentery. She died before the end of the month.

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(11) Mrs. L., aged 25, no children. The tumour was a sarcoma of the left ovary. At the time of operation there were no adhesions nor secondary growth, but there was much free blood-stained fluid in the abdomen, and she died from extension of the disease three months later.

(12) Miss de G., aged 50. Papillomatous disease of both ovaries. In her case also there was ascites, one of the cysts had ruptured, and there were malignant growths in the sigmoid, mesentery, and the parietal peritoneum. She died a few days after operation.

(13) Mrs. B., aged 64, two children. She had malignant disease of the left ovary. There was no other visible disease, but the abdomen was full of ascitic fluid, and she died of recurrence ten months after operation.

(14) Miss F., aged 42. Had compound papillary growths of both ovaries, and there was generalized malignant disease of the abdominal organs. She lived four and a half years after the first operation, and I am informed that she had two subsequent abdominal sections in the hope of removing the malignant growth. In this case the ovarian disease appears to have been secondary to a small scirrhous in the left mamma.

(15) Mrs. R., aged 51, one child. This seemed to be a particularly bad case of malignant papillomatous disease of both ovaries. There was free fluid in the abdominal cavity, many dense adhesions, and papillomata were disseminated all over the abdomen and pelvis, but she is alive and well (January, 1910) more than five years after operation.

(16) Mrs. H., aged 30, one child. Adeno-carcinoma of the right ovary. This was an interesting case. Patient came into hospital nearly at the full time of pregnancy; the pelvis was blocked by an apparently soft, solid tumour, which was thought to be a fibroid, and it was evident that delivery would have to be by Cæsarean section. The day before the time appointed for operation the patient had a sudden collapse which, when the abdomen was opened, was found to be due to hæmorrhage from the tumour; the cause of obstruction was not fibroid, but ovarian disease. Patient did well at the time, the uterus and left ovary appeared to be normal, but she died two years later with the abdomen full of recurrent growth.

(17) Miss B., aged 38. The operation in this case was undertaken for large multiple fibromata of the uterus. The ovarian cyst, which lay back and was not detected until the abdomen was opened, looked perfectly simple, but under the microscope its walls showed plaques where the epithelium had proliferated into the lumen. It was a tumour the size of a football, the inner surface of which was studded with nodules varying in size from that of a pea to 1 in. in diameter. The patient was a servant at the Royal Free Hospital, but unfortunately left the service and was lost sight of.

(18) Miss H., aged 45. On opening the abdomen papillomatous masses were seen growing from both ovaries, tubes, uterus, and intestines. Nothing was removed, but the patient survived the operation one year.

(19) Mrs. F., aged 32, no child. She had adeno-carcinomata of both ovaries. The growth had disseminated widely; the abdomen was distended by a moderate amount of fluid and quantities of solid growth; as much was cleared away as seemed possible. Patient died four months later. In this case the primary growth was probably in the stomach. The ovarian tumours were solid and looked like fibroids, containing solid nodules of growth.

(20) Mrs. T., aged 27, one child. In this case there was adeno-carcinoma of both ovaries. At the time of operation both cysts were almost filled with soft, yellowish, lobulated growth, which under the microscope was seen to be adeno-carcinoma; all the other organs appeared to be normal. Unfortunately there was no subsequent history.

(21) Miss M., aged 48. On opening the abdomen adeno-carcinoma of both ovaries was found, with widely-disseminated growths in the abdomen and pelvis. She died from extension of the disease four months later.

(22) Mrs. S., aged 23, one child. At the operation a smooth, non-adherent tumour occupied the position of the left ovary; no other growth was evident. On microscopical examination the disease was found to be round-celled sarcoma. Mrs. S. died seven months later with very abundant sarcomatous disease involving the uterus, right ovary, bladder and pelvic cavity.

(23) Miss W., aged 56. Sarcomata of both ovaries. This was a very interesting case. The uterus was removed for what appeared to be an ordinary fibroid in August, 1906; the ovaries at that time looked perfectly healthy and were left. The patient was not seen again until December, 1907, when she had two large ovarian tumours, and growths disseminated over the peritoneum, intestines, mesentery, and liver. The patient said that the tumours had appeared many months before, but that her Christian Science friends advised her not to have recourse to surgery, in the hope that faith and prayer would relieve her of her trouble. Unfortunately, her hope was disappointed, and she died a few days after the too-long-delayed operation.

(24) Mrs. F., aged 49, no children. Papilliferous adeno-carcinoma of the right ovary, complicated with multiple fibroids. The interesting point in this case was that there was a large cystic tumour with a cauliflower-like growth projecting into its cavity. Attached to the wall of the cyst there were solid papillary masses, and some of the individual growths were from 2 in. to 3 in. in length.

(25) Mrs. M., aged 55, one child. The patient had suffered from scirrhus of the right mamma, which had not been operated on. The duration of this disease was uncertain, and during the fourteen months that she was under observation it appeared to make no progress; the abdomen, however, gradually increased in size, and the quantity of fluid was eventually so great that operation became necessary to afford some relief to the respiration and circulation. Malignant disease was found generally disseminated in the form of ovarian growths and also disease of the surface of the stomach, liver, and intestines.

On looking over these cases one point that comes out is the comparatively advanced age of the patients, for, although of the 25 two were under 25 and three were between 30 and 35 years of age, the average age is rather over 46 years, whereas the average age of the non-malignant cases is just over 35 years. Another point to be noticed is the immediate mortality (by which I mean all deaths occurring within one month of operation), for whereas in the malignant cases it works out at 20 per cent., two only died of the 125 apparently innocent cases.

With regard to the question of whether malignant ovarian growths are primary or secondary it is extremely difficult to offer any opinion. Of the 25 cases at present under review, 4 were certainly secondary—Nos. 7, 14, 23, and 25—and No. 19 was probably secondary. In Case No. 7 adeno-carcinoma of both ovaries was secondary to cancer of the stomach; in Nos. 14 and 25 it was secondary to scirrhus of the mamma; in No. 23 to a uterine tumour that had been supposed to be fibroid, but was undoubtedly sarcoma; and in No. 19 it was probably secondary to cancer of the stomach. It is not, however, possible to say in how many of the remaining 20 cases the malignant ovarian growths were primary. In the absence of preceding symptoms and in the absence of any evident pathological condition of another organ revealed at the time of operation, nothing short of a very thorough post-mortem examination could determine whether or no a primary focus might have existed in the mammae, stomach, intestines, or elsewhere.

The more we study our cases the more is the conviction pressed home that it is necessary to submit all specimens to a careful microscopic examination by an expert pathologist, even those that appear perfectly innocent. To quote one example only, No. 4, Mrs. A., there was nothing in the appearance of her ovarian cysts to suggest malignancy, and yet only eight months elapsed before her pelvis was full of malignant growth; and again in No. 23, Miss W., the uterine tumour removed so closely resembled the naked-eye appearance of an ordinary fibroid that her brother, an experienced medical man, agreed with me that it was not worth while to have the tumour examined, and also that the ovaries, which seemed to us to be perfectly healthy, had better be left. Probably had we examined the tumour removed the sarcomatous nature of the disease would have been revealed, and an early ovariectomy might have prolonged or saved her life.

The importance of the subject is very great, for scientifically much might be learnt as to the nature and mode of growth of various forms of malignant disease; secondly, the prognosis of our cases would be more

accurate and therefore less likely to lead to disappointment ; and, thirdly, of the greatest practical importance, we could do more for our patients if we knew the nature of their trouble. At the present time there is a very great and lamentable waste of material both in private and in hospital work, and there is reason to fear that this will continue until the pathological branch of the profession can insure more adequate remuneration than it does at present. The pathological department in our hospitals is nearly always undermanned and badly starved in equipment. The recent action of the London University in laying stress on pathology as one of the chief subjects for examination shows that better days are coming, and it is to be hoped that many wealthy men will follow the example of Mr. Beit and come forward to endow laboratories and to make pathological research not only the honourable profession that it certainly is, but to insure that honour shall not be the only reward.

In conclusion, I must say a few words about classification. It is not until we turn over our cases with a view to writing a paper, and spend hours in an attempt at classifying them, that we sadly realize the difficulties to be encountered. There seem to be almost as many systems of classification as there are authorities, and the danger of cross-classification is great—e.g., a given growth may be at the same time multilocular, adenomatous, and carcinomatous ; another growth may be described both as a papilloma and as a malignant tumour. Might one not venture to suggest that in the near future a committee of pathologists might be formed who should endeavour to provide us with a really scientific system of classification—one that should make researches and the writing of papers much more of a real joy than it is at present ?

Details of Twenty-five Malignant Cases of Ovarian Growths.

No.	Name	Date	Age	Parity	Nature of tumour	Primary or secondary	Condition found at time of operation	Subsequent history	Remarks
1	Mrs. E.	Oct. 10, 1892	52	—	Papillomata, both ovaries	—	Disease invading bladder, &c.	Died	—
2	Miss D.	July 11, 1893	54	—	Papillomata, both ovaries	? Primary	One small papilloma on the parietal peritoneum	Died one year after of generalized cancer of peritoneum	—
3	Mrs. M.	July 24, 1893	49	3	Sarcoma, both ovaries	—	All apparently normal	Died of sarcoma uteri, which appeared one year after ovariectomy	—
4	Mrs. A.	August 3, 1893	45	3	Adenoma, both ovaries	—	All apparently normal	After eight months disease sprouted through lower angle of cicatrix and filled pelvis	—
5	Miss B.	Jan. 15, 1895	35	—	Papillomata, both ovaries	—	Disseminated growths in abdomen.	No subsequent history	—
6	Mrs. G.	March 26, 1895	33	1	Papillomata, both ovaries	—	Ascites, dense adhesions	No subsequent history	—
7	Mrs. T. S.	Feb. 5, 1896	40	2	Adeno-carcinoma, both ovaries	Secondary	Both ovaries freely movable; solid, white, dense; no hint of other disease	Secondary to cancer of stomach; died of intestinal obstruction ten months later	—

8	Miss P.	March 13, 1897	64	—	Malignant ovarian cysts, both ovaries	—	Malignant disease, omentum and peritoneum	Died about two months after operation	—
9	Miss Co.	Feb. 12, 1899	69	—	Colloid cancer	—	Abdomen full of fluid and colloid material, which had escaped from a ruptured cyst of the left ovary; no other growth seen	Died on twelfth day	—
10	Miss Ch.	June 30, 1899	68	—	Papillomata, both ovaries	Primary	Free fluid in abdomen; secondary growths in colon, mesentery and omentum	Died	—
11	Mrs. L.	Oct. 10, 1899	25	—	Sarcoma, left ovary	—	Much free blood-stained fluid	Died from extension of disease three months later	—
12	Miss de G.	Oct. 13, 1899	50	—	Papillomata, both ovaries	Primary	Ascites, ruptured cyst, growths in sigmoid, mesentery and peritoneum	Died	—
13	Mrs. B.	June 23, 1900	64	2	Malignant cyst, left ovary; papillomata	Primary	Ascites	Died ten months after operation	—
14	Miss F.	Dec. 9, 1903	42	—	Compound malignant papillomata, both ovaries	Secondary	Fibroids of uterus; both ovaries papilomatous masses; large, solid, malignant mass in mesocolon	Lived four and a half years from first operation; had two subsequent abdominal sections	Secondary to small scirrhous in left mamma
15	Mrs. R.	Nov. 2, 1904	51	1	Malignant papillomata, both ovaries	Primary	Many adhesions and disseminated papillomata	Alive and well January, 1910, five years later	—

No.	Name	Date	Age	Parity	Nature of tumour	Primary or secondary	Condition found at time of operation	Subsequent history	Remarks
16	Mrs. H.	Jan. 19, 1905	30	1	Adeno-carcinoma, right ovary	Primary	Complicating pregnancy at term; Cesarean section; uterus and left ovary apparently normal	Readmitted, April, 1906; tumour of left ovary, similar to that of right, smooth, partly cystic, 6 in. in diameter; died Sept., 1906; post mortem, a few small metastatic nodules in lower abdomen, none elsewhere	—
17	Miss B.	Sept. 21, 1906	38	—	Adeno-carcinoma, left ovary	—	Complicated with multiple fibroids	No subsequent history	The ovarian cyst looked quite simple, but under microscope showed proliferation of epithelium into lumen; it was a large tumour, the size of a football, the inner surface was studded with nodules varying in size from that of a pea to 1 in. in diameter
18	Miss H.	Sept. 24, 1906	45	—	Papillomata	—	Generalized; ovaries, tubes, uterus and omentum	Survived exploratory operation one year	—
19	Mrs. F.	Nov. 29, 1906	32	—	Adeno-carcinoma, both ovaries	Probably secondary	Whole abdomen full of solid growths	Died after four months	Primary growth probably in stomach; the macroscopic appearance of the ovaries points to this; they are solid, fibroid-like tumours, containing scattered nodules of growth

20	Mrs. T.	May 3, 1907	97	1	Adeno-carcinoma, both ovaries	Primary	All appeared normal	No subsequent history	Both cysts almost filled with soft yellow lobu- lated growth; micro- scope showed "definite adeno-carcinoma"
21	Miss M.	Sept. 21, 1907	48	—	Adeno-carcinoma, both ovaries	—	Disseminated growths in pelvis	Died four months later	—
22	Mrs. S.	Oct. 10, 1907	23	1	Round-celled sar- coma, left ovary	—	—	Died seven months later; extension to uterus, right ovary, bladder, and pelvic cavity	—
23	Miss W.	Dec. 16, 1907	56	—	Sarcoma, both ovaries	Secondary	Growths all over pelvis, mesentery, intestines, liver	Died	Operation for supposed fibroid in August, 1906; tumour soon appeared but was neglected; patient seen in <i>extremis</i> , December, 1907
24	Mrs. F.	May 8, 1908	49	—	Papilliferous adeno-carcinoma, right ovarian cyst	?	Complicated with multiple fibroids	No subsequent history	Large cystic tumour, cauliflower growth pro- jecting into cavity, also solid masses adherent to the wall; some of the growths measured 2 in. to 3 in. in length
25	Mrs. M.	June 8, 1909	55	1	Papillomata	—	General: dissemina- ted over stomach, liver and intestines	Secondary to scirrhus of right mamma; died one month later	—

DISCUSSION.

The PRESIDENT (Dr. H. Macnaughton-Jones), in inviting discussion, said that the Section was indebted to Mrs. Scharlieb for a paper which had an important pathological and clinical interest for all. The difficulty in arriving at a prognosis as to future recurrences or metastases was frequently great, and they were still in considerable doubt as to the nature of the changes which converted certain apparently benign cystomata into malignant.

Dr. AMAND ROUTH congratulated Mrs. Scharlieb on her valuable paper, and agreed with her as to the difficulty of classifying papillomata of the ovary, especially with regard to their malignancy. He believed that almost all varieties of papillomata developing on the inner surface of an ovarian cyst are liable to take on a malignant character when they sprout through the cyst wall and reach either the peritoneum or the connective tissue. In some cases Mrs. Scharlieb had used the word "papilloma" in a non-pathological sense, from the naked-eye appearance only. Thus ovarian papillomata in two cases were stated to have been secondary to scirrhus of the breast. His own cases showed 10 per cent. of true cancer, 4 per cent. of malignant papilloma, and 6 per cent. of so-called benign papilloma—that is, papillomata which were still confined to the cavity of the ovarian cyst—and 16 per cent. were dermoids. As showing how curiously cases run together, he mentioned that in the last 16 cases of ovarian tumour operated on by him at the Charing Cross Hospital, 7 were true cancer and 1 was a malignant papilloma. He hoped that some generally recognized classification of ovarian tumours would soon be arrived at, especially as regarded the question of their malignancy.

Mr. GLENDINING said that he should confine what he had to say to the pathological side of the question. In considering the 25 malignant cases presented, he thought those occurring before 1904 were practically useless, as they were operated on at a late stage of the disease and as the diagnosis was not in conformity with our present knowledge, and, finally, it was of little use having results, if what they were the results of was unknown. The classification adopted for these cases was a somewhat meaningless one. On broad lines the classification of ovarian growths into three distinct groups was not a difficult matter—viz.: (1) Simple cysts, new growths, and cyst-adenomata; (2) malignant growths; (3) the papilliferous cyst-adenomata, comprising a semi-malignant class which, in their mode of growth and also in their histological appearances, differed very distinctly from true carcinoma. This last group never killed by malignancy, but simply as an accident of growth. He offered some figures on the relative malignancy of ovarian tumours taken from the pathological records of the Chelsea Hospital for Women for the years 1908 and 1909. There had been examined during the last two years 106 cases of ovarian tumours (lutein, follicular cysts, and tubo-ovarian cysts were not included). The relative

malignancy was 17 per cent. (11·3 per cent. primary carcinoma and 5 per cent. secondary carcinoma). The primary malignant cases were composed of columnar or cubical-cell carcinoma, 5·7 per cent.; endothelioma, 1·8 per cent.; and sarcoma, 3·8 per cent. The cases of secondary carcinoma were, as might be more readily understood from the following facts, divisible into two groups. He recently examined the cancer register of the Middlesex Hospital with the idea of ascertaining the relative frequency of secondary carcinoma of the ovary, and found that the incidence upon that organ for all cases of carcinoma in a series of 690 post-mortem examinations was 5 per cent. These secondary carcinomata of the ovary were readily divided into two classes occurring in equal frequency—i.e., 2·5 per cent. of all cases: (1) Those arising as the result of direct extension from neighbouring viscera, colon, rectum, uterus, &c.; (2) those the result of dissemination from a distance via the peritoneal cavity, and invariably of the spheroidal-celled type of carcinoma and secondary to that of the breast, stomach, intestine, or liver. These facts were well borne out in the instance of the cases of secondary carcinoma examined in the Chelsea Hospital statistics. A noteworthy fact, and one to which little attention has been drawn, is the frequency with which secondary carcinoma is engrafted upon a pre-existing cyst-adenoma, and which is recognizable as distinct localized areas of white compact growth, striking by contrast with the typical cyst-adenomatous structure. He had personally encountered 4 cases during the last year. Finally, in order that statistics of the relative malignancy of ovarian tumours should have any real value, it was essential that there should be some unification of classification founded upon a serious histological examination.

Dr. GRIFFITH referred to the difficulty of the exact diagnosis of many cases of ovarian tumour, and wished to ask Mrs. Scharlieb if the diagnosis in her 25 cases was from pathological as well as clinical characters. If not, he doubted whether there were not some cases included which were not malignant.

The PRESIDENT said he had gone through 100 cases of removal of the ovaries for various growths and degenerations. Out of this number there were 5 carcinomata, 3 of these being adeno-carcinoma, 1 sarcoma, 1 malignant papillary dermoid cyst, and one papillary carcinoma. One of the carcinomata was a giant scirrhous carcinoma of one ovary which filled the abdomen, and there was an adenomyoma of the other. All the remainder were benign, including two fibromata and two adenofibromata. Seeing that the paper was of a statistical nature, he had sought for and obtained the statistics from the foreign clinics of a few personal friends. He would like to read an excerpt from a letter from Professor August Martin (Berlin) on the subject: "True neoplasms are always of doubtful prognosis; until we have exact statistics of microscopical examination of every true neoplasm, a reply as to the relative frequency with which malignancy is found must have a limited value. Even with a modern typical scheme, and statistics collected in accordance with it, it would take ten years to answer the question." Professor Schauta (Vienna) reported, out of

334 tumours of the ovary, 77 malignant, including 32 adenoma and carcinoma, 17 sarcoma, 28 proliferating papillomatous cystoma. Professor Jacobs (Brussels) reported (between 1897 and 1910) 2,322 cases of cystic tumour of the ovary. Of these, 827 were proligerous glandular cysts, 102 vegetating cysts, 318 proligerous papilloma, 177 colloidal cysts, 189 epitheliomatous, 297 dermoid, 114 mixed (mucoid and dermoid), 289 parovarian, 41 fibroid of the ovary. Professor Winter (Königsberg), from December, 1905, to December, 1908, reported: of 112 tumours of the ovary, 18 were malignant. Professor Schottlaender, the distinguished pathologist of the late Professor Rosthorn's clinic in Vienna, whose lamented death last year was a great loss to gynaecology, sent an exhaustive report of 125 tumours which were operated on during the last year and nine months; of these 25 were malignant. It is worthy of note that, excluding retention cysts such as simple serous and lutein and follicular cysts, he did not include under the head of malignancy proliferating glandular cyst-adenomata, simple papillary cysts, or proliferating papillary cyst-adenomata, of which there were 60. There were 19 teratomatous tumours, and 16 dermoid. Fibroma of the ovary was present in three cases. Of the 25 malignant tumours, 18 were carcinomatous, of which 8 were of the papillary type. There were 7 cases of solid carcinoma, and 1 carcinomatous dermoid cyst. The 5 cases of sarcoma were divided thus: 3 fibrosarcoma, 1 round-celled, 1 perivascular; there was one case of endothelioma and one of hypernephroma. No case of secondary malignant growth of the ovary was included. Metastatic carcinoma of the brain occurred once, metastasis from cancerous stomach once. Professor Krönig (Freiburg), Professors Zweifel and Mangiagalli were compiling the statistics of their clinics, but they had not yet reached him. Analysing still further these 2,893 cases, the records of 1,045 show that 659 were unilateral, 386 bilateral. Of the entire number, 309 were indisputably malignant—a little over 9 per cent. Of the latter growths, 189 were epitheliomatous, degenerating cysts (not differentiated), and 18 under the head of "malignant" were not differentiated; 32 adeno-carcinomatous cysts; 28 proliferating papilloma; 22 sarcoma; 18 various types of carcinoma, as glandular and papillary adenoma, and cyst adenoma, solid malignant adenoma, and malignant dermoid adenoma; and 1 endothelioma. Of the 41 cases in which there is a record of the unilateral or bilateral occurrence, 26 were unilateral and 15 bilateral. He trusted that Mrs. Scharlieb might be pleased to add these statistics to those she had collected. With regard to primary or secondary growths, it was often extremely difficult to say whether a growth was primary or not, so frequently were the adjacent viscera involved, and occasionally some other abdominal organ. He quite felt, as Mrs. Scharlieb did, the desire for a more definite differentiation between the benign and malignant growths of the ovary. At present a good deal of confusion existed. He noticed that there was no case of malignancy arising from a dermoid in her list. One clinical feature that struck him in many of these cases was the absence of any marked pain; consequently the suspicion of malignancy was not aroused until the disease was far advanced.

Mrs. SCHARLIEB thanked the President and those members of the Section who had discussed her paper, and said that she feared that, after all, the definition of malignancy had advanced little further from the time of Pozzi, who said "malignancy is not a pathological entity, but a clinical phenomenon."

An Atypical Malignant Tumour of the Uterus.

By HERBERT J. PATERSON, F.R.C.S.

THE patient from whom this uterus was removed was single, and aged 43. She suffered from metrorrhagia, gradually increasing in severity, for six or seven months. The uterus was uniformly enlarged, and I thought that it was the seat of a soft, rapidly-growing fibroid. On cutting into the uterus after removal a large, red, very vascular tumour was found, gelatinous-looking in places, which I took to be a sarcoma. This view was confirmed by microscopical sections. The operation was performed in January, 1904, and on December 20, 1909, I heard that the patient has remained perfectly well ever since the operation, and has had no symptoms of recurrence. A few weeks ago I had the specimen reopened and fresh sections cut. I think that the exact nature of the tumour is doubtful. There are places in the sections, especially the more recent section, which resemble a carcinoma. The size of the tumour, and the fact that, notwithstanding its rapid growth, there was no glandular involvement, are against its being a carcinoma. The vascularity of the tumour and the areas of degeneration are in favour of sarcoma. There was no unusual feature about the operation, but had I known that the tumour was malignant I should of course have performed a panhysterectomy instead of a sub-total hysterectomy; but as the patient has remained well for six years, the result of the simpler procedure may be considered very satisfactory.

Report of Pathology Committee.—The Committee consider the growth to be an atypical one of the uterus. It is not a sarcoma. The bulk of the tumour is alveolar in structure, resembling carcinoma, but whether its origin is from endo- or epithelial cells it is impossible to determine.

DISCUSSION.

Dr. CUTHBERT LOCKYER thought that the thanks of the Section were due to Mr. H. J. Paterson for bringing forward such an interesting case; he had studied the sections and examined the tumour. The histology of the growth presented many features of a carcinoma—e.g., the alveolation, with cells contained in alveoli, apparently without the intervention of intercellular stroma. Again, the character of the cells was not unlike that of a spheroidal-celled carcinoma; on the other hand, there were tracts showing vessels from the walls of which cell-proliferation was proceeding, and where there could be seen degeneration areas like those common in endotheliomata. Dr. Lockyer was disposed to regard the growth as an endothelioma, and the long post-operative history upheld this diagnosis. He cited a case shown by Mr. Doran in which—although the cervix had not been removed, and although the growth (a perithelioma) had spread to the tube and invaded the ovary and round ligament, and had also formed a deposit in the broad ligament the size of a man's fist—the woman was, nevertheless, alive and well four years after operation. The case was obviously one to be submitted to the Pathology Committee.

The PRESIDENT suggested that, in face of the divergence of opinion as to the pathological condition, the specimen should be referred to the Pathology Committee, and this was done.

Case of Adenoma Malignum Cysticum Cervicis Uteri.

By JOHN BENJAMIN HELLIER, M.D. (Leeds).

SUMMARY: A woman was admitted to hospital with extensive malignant growth in vagina of more than eighteen months' standing. The growth sprang from the cervix and filled the vagina without involving it. It was treated by preliminary scraping, and then by abdominal panhysterectomy, with recovery. The growth was found to be malignant adenoma with very extensive formation of small cysts.

The patient, aged 46, had been married at the age of 19, and had had eleven confinements without any serious complication. For a year and three quarters before her admission she had suffered from almost continuous blood-stained discharge, which of late had been increasingly foul, with frequent attacks of flooding. She said that twelve months

before her admission she had been discharged from a hospital in another town, after a slight operation, as incurable. On admission to the Leeds Infirmary on September 16, 1909, she presented a profoundly anæmic appearance, her complexion being strikingly wax-like. There was a fœtid vaginal discharge and some degree of pyrexia. She was thin, but not greatly emaciated. On examination the vagina was found to be filled with cauliflower-like growth, necrotic and foul. When this was scraped away it was found that the vaginal walls were not involved, and that the uterus was quite mobile and not much enlarged. The other organs appeared to be healthy. As her condition at that date seemed too bad to permit a radical operation, she was sent to our convalescent home, from which she returned in three weeks in a greatly improved condition. On October 23 abdominal panhysterectomy was performed,

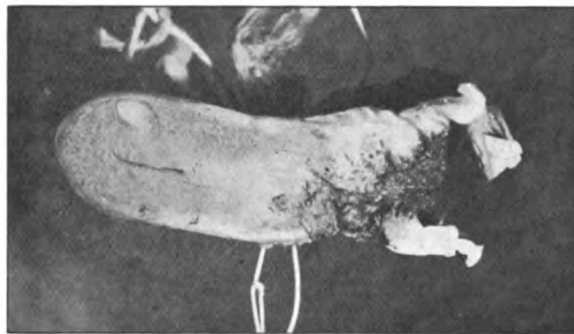


FIG. 1.

Adenoma malignum cysticum. Photograph of the uterus.

the ureters being drawn aside and the vagina clamped according to Wertheim's method. No cancerous glands were found. The growth was perforating the cervix at a point opposite the os internum in the anterior wall, as seen in the specimen, and the bladder wall tore a little at this spot during separation from the uterus. Fortunately there seemed to be little infiltration of the bladder wall here. The rent was closed with catgut and the patient made a good recovery, the bladder healing without any leaking and the abdominal wall with only one small point of suppuration. She went out wonderfully improved in health, and has done well up to date.

I show the uterus as removed by operation, also a photograph of the uterus (fig. 1); also a microscopic section of the growth in the

cervix, with microphotograph (fig. 2). These were made for me by Dr. O. Grüner, of Leeds.

The uterus shows the ragged cervix below left after the polypoid mass was scraped away at the preliminary operation. A ring of vaginal wall is attached to the cervix. It should be said that at the hysterectomy a further piece of the vagina was removed on the left side to make sure of a healthy margin at that point. A longitudinal section has been made through the specimen, and the growth can be seen infiltrating the cervix up to about the level of the os internum. Here it perforates the anterior wall. All over the infiltrated cervix are



FIG. 2.

Microscopic section of the growth in the cervix.

seen gland spaces, visible to the naked eye and very well seen in the lantern slide. In the recent state they looked like sago grains. On microscopic section they are seen to be large cysts lined with low columnar or cubical epithelium. There is usually only one layer of cells, and there is very little infiltration of the fibrous stroma. The section resembles the structure of the more solid parts of an ordinary proliferating ovarian cyst of benign nature. Although the malignancy of the tumour is beyond doubt, yet, considering the length of time during which it remained operable, and the mobility of the uterus, and the

small amount of infiltration into surrounding parts, it would seem to be of a relatively low degree of malignancy.

I find this kind of growth described in Veit's "*Handbuch der Gynäkologie*," 1897, iii., 2 Hälfte, p. 224, and a very interesting case is given in detail in the *Zeitschrift f. Geburtshülfe*,¹ in a paper by Knauss and Camerer. Here, too, there was a polypoid vaginal growth with an infiltrated cervix, and the microscopic sections closely resemble those from my case. This patient was treated by vaginal hysterectomy, and was found free from recurrence ten years later. It is from Knauss and Camerer that I take the term *adenoma malignum cysticum*.

I am sorry that distance and various engagements prevent me from reading my case myself before the Section to-night, and I desire to thank Dr. Taylor, who kindly takes my place.

Report of Pathology Committee.—The Committee consider Dr. Hellier's description of the tumour to be correct.

Dr. CUTHBERT LOCKYER pointed out that neither in the sections shown nor in the microphotograph was there any evidence of malignancy. The sections presented the structure of simple dilated gland-spaces. He suggested that more tissue should be cut from the cervix at the level of the internal os, and also from the cauliflower excrescence which had been removed, and that these fresh sections should be sent to the Pathology Committee for report.

Myomatous Tumour of the Uterus simulating Sarcomatous Growth.

By H. MACNAUGHTON-JONES, M.D.

THE President showed a myomatous uterus in which the growths macroscopically closely resembled sarcomata growing from the fibromuscular wall of the uterus. The patient was aged 42, and had had her last pregnancy seven years before the operation. She had erratic menstruation and a foul-smelling discharge, and naturally there was a suspicion of malignancy, as a smooth, dark-red, polypoid bleeding growth protruded from the os. On examination this was found to grow from a

¹ *Zeitschr. f. Geburts. u. Gynäkol.*, Stuttg., 1896, xxxiv, p. 446.

wide base on the right side of the uterus, and there was obviously a large solid growth involving the body and the cervix at the right side. Panhysterectomy was performed, and the patient did well. As there was vomiting, which began on the third day after operation, she had proctoclysis, and varying quantities of saline, at the rate of 2 pints in the four hours, were administered on and off for three days. On macroscopical examination the sessile polypoid growth was found to spring from the right lateral wall of the uterus, and to be an offshoot from a projecting myomatous mass; and in the cavity at the fundus there was another suspicious, soft, submucous growth sprouting into it. Dr. Cuthbert Lockyer's report, however, which was as follows, came as a relief:—

“The larger tumour is a fibromyoma in which the component parts take up the staining reagents thoroughly well. It is built up of muscle bundles, held together by a relatively small amount of connective tissue. It is covered by the mucosa of the cavum uteri, from which a few tubules dip into the superficial and muscular strata. The smaller submucous growth was of a very soft consistence before dehydration. It shows a surface of hypertrophied mucosa, whilst the tumour itself is undergoing hyaline and fibroid degeneration. The hyaline fibrous tissue exists in whorls, which include and press on the muscle tissue, thereby causing a diminution in the amount of the latter. Both tumours are quite devoid of malignant characteristics (sarcomatous or otherwise).”

Prolapsus Uteri with severe Cardiac Disease operated on under Local and Spinal Analgesia.

By J. INGLIS PARSONS, M.D., and T. C. CLARE, F.R.C.S.

THE patient, aged 39, was admitted to the Chelsea Hospital for Women on January 2, 1909; she had had three children, and her last child was born five years previously. Her menstruation had been regular, but of more than normal amount. She suffered for some years from a profuse white and yellow vaginal discharge. The bladder was healthy and had given no trouble, and her bowels were regular. She complained of bearing-down pain, severe pain in the sacral region, and protrusion of

the womb. The prolapse began eight years ago, and after the birth of her third child, five years ago, the uterus became procident. During the last two years several varieties of pessaries had been tried, but failed to keep up the uterus.

Examination of the heart revealed displacement of the apex to the fifth space $4\frac{1}{2}$ in. from the middle line; enlargement of the left ventricle; a loud systolic mitral bruit, and also a loud systolic and diastolic aortic bruit. The pulse was fairly good, but on the weak side faster than normal, sometimes irregular, with the typical water-hammer character associated with aortic regurgitation. On the other hand, her nutrition was good, and she was, if anything, on the plump side, with a good colour, showing that the valvular disease was well compensated.

A pelvic examination revealed a procident uterus, with enlarged cervix and extensive erosion. The perineum was torn through to the sphincter ani, and both vaginal walls protruded 1 in. or more. Although at first I refused to do anything because I did not think she could take an anæsthetic without grave risk, I decided to try local analgesia and injection of the broad ligaments for the prolapse. She was kept in bed for ten days and the heart toned up. On January 12 the first operation took place. While in the lithotomy position, 1 dr. of saline solution containing $\frac{1}{8}$ gr. of eucaine B was injected into each broad ligament. After waiting five minutes, 80 m of my quinine solution were injected into the right broad ligament. It caused a great deal of pain and she became distinctly faint; the pulse could hardly be felt. An injection of strychnine was given at once. After waiting ten minutes she recovered herself, and the pulse showed great improvement. A second injection was then made at her own express wish into the left broad ligament. This caused almost as much pain and faintness as the first injection, but she recovered from it more quickly. After her return to the ward all her pain was gone, and she also remained free from pain afterwards. There were no post-operative troubles, and she left the hospital on February 6 wearing a metal pessary. I did not expect a very good result because the injection of eucaine would dilute the quinine solution and probably interfere with the reaction. However, at the end of five months she came up to the hospital, and the uterus was found to be in good position and well held up, and she was very much improved and able to do without the pessary. She still felt a good deal of discomfort from the ruptured perineum, and so I readmitted her on May 20, 1909, with a view to doing a colpoperineorrhaphy. She was kept in bed for five days and given liquor strychnia 5m and tinctura strophanthi

5 m t.d.s. I suggested that she should have the operation under spinal analgesia on account of the condition of her heart, and pointed out to her the extra risk involved. She embraced the proposal with avidity.

On May 25 the injection was made by Mr. T. C. Clare, house surgeon to the hospital, the patient sitting on the operating table with her back bent well forward; 1 c.c. of a 5 per cent. solution of tropacocaine in 6 per cent. sodium chloride solution was injected into the spinal theca between the third and fourth lumbar vertebræ. She was then laid on the table, with the shoulders raised a little by pillows to limit the rapidity of the upward diffusion of the solution. After five minutes she was placed in the lithotomy position, and analgesia was found to be satisfactory. The patient said she could tell something was being done, but felt no pain whatever. Throughout the operation of colpoperineorraphy, which lasted about twenty minutes, the pulse maintained its ordinary rate and there was no evidence of cardiac distress. The patient was not in the least disturbed by the operation, and chatted to the matron the whole time.

Two hours after the operation she felt faint, and actually fainted shortly afterwards. This was probably due to the fact that the head of the bed had been raised 8 in. as an additional precaution against any upward diffusion of the tropacocaine. She recovered as soon as the bed was lowered, and there was no recurrence of faintness.

Remarks.—It has been said that advanced cardiac disease constitutes a contra-indication to spinal analgesia. This case is of interest as showing that the method may be employed for operations on the pelvis, even where severe valvular lesions exist.

Pelvic Hæmatocele of Ovarian Origin.

Shown by CUTHBERT LOCKYER, M.D.

Mr. STANLEY BOYD has kindly given me the details of two interesting cases upon which he operated for supposed acute appendicitis, but which proved to be cases of pelvic hæmatocele in which there was no evidence of pregnancy. In both instances the Fallopian tubes were intact, pervious, and undamaged; in both an ovarian blood-cyst had

ruptured and was the cause of the internal bleeding. To these two cases of ovarian hæmorrhage, not due to gestation, I add a third (by the kind permission of Dr. Amand Routh) in which, after the left tube had been removed for ruptured ectopic gestation, a pelvic hæmatocele formed on the right side, due to ovarian bleeding. In this case and in one of Mr. Boyd's two cases I have sought by microscopic investigation to find proof of ovarian gestation, but no evidence thereof has been obtained. The details supplied by Mr. Stanley Boyd are as follows:—

CASE I.

A young woman, previously in good health, was taken ill on September 21, 1908, with diarrhœa and abdominal pain. This began in the morning and got worse by night; she was seen at 10 p.m. by Dr. Bone, of Luton, who found her suffering from abdominal pain, with slight general distension and tenderness. The pulse-rate was 100 and the temperature 99·5° F. On September 22 (next day) the patient went through the ceremony of marriage at 10 a.m. and took part in the "breakfast"; she went to bed at 5 p.m. and in the evening her pain was worse. The pulse-rate had increased to 120 and the temperature reached 100° F. On September 24 there was tenderness all over the abdomen, and distension; pulse 132, temperature 100° F. There was nothing to be made out *per rectum*. On September 25 Mr. Boyd saw her and thought she probably had appendicitis. He incised the abdominal wall outside the right rectus and a little clear fluid escaped, which had nothing to do with the appendix. Then, after making a paramedian suprapubic opening and separating adherent coils of gut, Mr. Boyd, to quote his own expression, "found himself in a hæmatocele." Of course, ectopic gestation was thought of and also the patient's refusal to postpone marriage. Dr. Bone, however, did not think this diagnosis likely. The clot indicated the left side as the source of the hæmorrhage.

The tube was red (probably stained) and the ampullary end somewhat swollen, the orifice was patent but not large, there was no blood issuing nor to be squeezed from it. No trace of a foetus could be found. The left ovary was represented by a thick walled ($\frac{1}{2}$ in.) ruptured and collapsed cyst. This was adherent far back on the left side of the pelvis and surrounded by clot. It was removed, as was also the right ovary, which was a mass of small cysts. The patient made an uninterrupted recovery.

CASE II.

A single young woman was seized with pain in the right iliac region at 1 a.m., June 29, 1909, whilst attending a ball. This pain she attributed to a slip earlier in the evening. She danced several dances after this and got to bed about 4 a.m., but the pain kept her awake until 7 a.m. She went to church at 11 a.m. feeling much better, but during the service the pain recurred badly. She went home and Dr. John Harold was sent for. He found her looking very ill, with a rapid, feeble pulse and much tenderness over the right iliac region. Dr. Harold sent the patient to a home, where she arrived at about 3 p.m., and soon after admission the pulse-rate was 120 and the temperature 99° F. Mr. Boyd saw her at 5.30 p.m., looking "pretty well, but wriggling about with pain." The pulse-rate was 126. The abdomen was not moving, but soft, markedly tender over the appendix region, which was also the seat of pain. There had been no vomiting. No pelvic examination was made. Mr. Boyd and Dr. Harold agreed that an early appendicitis was the most probable diagnosis, and at 6.30 p.m. the abdomen was opened in a line external to the right rectus. A little bloody fluid ran out and a mass of clot could be felt in the position of the right ovary. Through a paramedian suprapubic incision about a pint of recent clot was removed. The right ovary was adherent to the pelvic wall in the normal position, with a lot of clot about it. On its anterior free surface there was a rent in the wall of a cyst, having a diameter equal to that of a florin. The tube was apparently normal. The left ovary contained a cyst the size of a cob-nut. This was opened and its lining removed. Easy recovery followed. This patient had been under treatment for dysmenorrhœa, her periods having been slight, painful, lasting three or four days, and regular. The last period ended one week before this hæmatocele formed.

The specimen in this case was given to me to examine, and, on microscopic section, it is seen that the ovarian stroma contains a zone of interstitial hæmorrhage around the wall of the ruptured cyst. The cyst itself contains blood-clot, and in the latter lie segments of the lutein convolutions of a retrograde corpus luteum, and I regard the histological findings as proving that the hæmorrhage proceeded from a corpus luteum which had matured at the last menstrual epoch—i.e., one week prior to operation. In an article on the "Corpus Luteum and Luteum Cysts" which I read before the parent Society of this Obstetrical Section in 1905,

and which appeared in the *Transactions of the Obstetrical Society of London* (1905) 1906, xlvii., p. 164, I mentioned, under the description of "Lutein Hæmatomata," that these blood-cysts were formed in the pre-dehiscent stage of a Graafian follicle. They show no sign of the presence of lutein convolutions such as are seen in a matured corpus luteum; their tendency is not to rupture, but to shrink and become absorbed, or else to form a fibrous node, or, if infection occurs, they may cause a lutein abscess, or, finally, they may calcify. In this specimen of Mr. Boyd's the case is quite different: the opposite ovary contained no corpus luteum. There must have been one present, which ripened and discharged an ovum within a few days of operation. The only compact lutein tissue to be found is that seen in the blood-clot of the ruptured cyst, and the unavoidable conclusion is that this torn cavity represents the recent corpus luteum. I referred to the possibility of a retrograde corpus luteum behaving in this way in the above-named article, expressing the opinion that such cases must be extremely rare, and adding that Mr. Targett had made a microscopical investigation in a case which only admitted of this interpretation.

CASE III (DR. ROUTH'S).

A married woman, aged 28, was admitted into the Golding Ward of Charing Cross Hospital and an emergency operation was performed on the evening of July 22, 1905, by Dr. Routh, assisted by myself. At the time of operating the patient was collapsed from repeated recent hæmorrhage. The abdomen contained a large quantity of fluid blood, the left tube was ruptured in its outer half, and adherent to it was a mass of blood-clot. The ruptured tube and adjacent ovary were removed. A smooth convalescence followed. This woman was again admitted on December 5, 1906. She then complained of pain in the right groin. She had been married eleven years, had three children, aged $10\frac{1}{2}$, $8\frac{1}{2}$ and 3 years; no miscarriages; had thrice been in hospital before, in 1905 for left ectopic gestation (*vide supra*, p. 107). Menstruation began at the age of 11; regular in time, lasted seven days. The patient had been quite well since her last admission in July, 1905, up to November 18, 1906, when a "period" started in the morning. In the evening of the same day violent pain in the right groin set in, and this persisted until the evening of the next day (November 19). In character the pain was boring and localized, it did not shoot nor radiate, and was considerably

relieved by fomentations. The menstrual flow ceased at 4 o'clock on the day it began, but was again resumed on November 21—five days later. The loss was slight but foul-smelling, and continued until November 26. The boring pain gave way to a dull ache, which lasted until admission on December 5. Defecation and micturition caused general abdominal pain, and “fainting and giddiness” had been complained of. Dr. Routh found the uterus anteverted and there was a round hard body, the size of a cricket-ball to its right in the pouch of Douglas. On December 10, 1906, Dr. Routh, assisted by Dr. Eden, opened the abdomen and found the pouch of Douglas partly filled with blood-clot, whilst the right ovary was also distended with clot and presented a large rent on its surface. The tube was intact, its mesosalpinx normal, whilst the fimbriated end was closed.

The case was regarded as one of ovarian gestation and the parts given to me to examine. I cut numerous sections of the wall of the ruptured cyst and also of the ovarian stroma, but failed to discover any traces of gestation.

In this case we have a woman who menstruated at her proper time and was seized with violent dysmenorrhœa a few hours after the onset of the flow, and in whom a ruptured ovary and pelvic hæmatoma were found three weeks later, and yet no proof of ovarian gestation could be obtained. The section of the ovarian cyst wall is far more instructive than those obtained in Mr. Boyd's case. They show the lutein convolutions of a mature follicle still *in situ*. The theca interna is sodden with blood and so is the ovarian stroma, which forms the external coat of the cyst. The convolutions themselves are broken up and teased asunder by free hæmorrhage, whilst engorged capillaries filled with red disks are seen lying in the delicate connective tissue which keeps the lutein convolutions together. I should mention that the clerk in writing up the notes of the operation described this cyst as a gestation sac, and it was only after careful microscopic search that I found to my disappointment that it was not so.

Such cases as the three now recorded are not mentioned in text-books, and Mr. Targett is the only observer who has told me that he is familiar with this physiological accident. In bringing them before the notice of the Section, my object is to ascertain if other Fellows and Members have met with similar experience.

DISCUSSION.

The PRESIDENT said that he thought it had been clearly shown for some years that a pelvic hæmatocele might occur independently of the presence of gestation, and the differentiation of the two classes—that which was of a gestation origin, and that which arose in the corpus luteum and was associated with lutein changes—he had embodied in his own last edition. For this more exact knowledge he was at the time indebted to the paper of Dr. Cuthbert Lockyer read at the Obstetrical Society. He had had cases which undoubtedly were not due to gestation.

Dr. ARTHUR GILES said that he had come across a case which presented some points of similarity to those described by Dr. Lockyer, and it was further interesting, inasmuch as the pathological reports on the cases were made by Dr. Lockyer. The patient, aged 28, presented herself at the Prince of Wales' General Hospital, Tottenham, in July, 1908, with symptoms pointing to extra-uterine pregnancy—that is to say, that she had missed a period, had been seized with pain in the right side, and had suffered thereafter from a brownish discharge. She was admitted, and at the operation free blood was found in the pelvic cavity, the left appendages were normal, and the right tube appeared also normal; but there was a clot attached to a rent in the right ovary. It was thought that it might be a case of ovarian pregnancy, and the specimen was sent to Dr. Eastes's Laboratories of Pathology and Public Health. The following report was made by Dr. Lockyer: "The blood-clot adherent to the ovary has been examined by taking a section through the ovary where the former is adherent to it. There is no sign of gestation present. The edges of the clot show the ovarian stroma arranged as it would be around a lutein hæmatoma. The free pieces of clot have not been examined." The case therefore appeared to be one of the same type as Dr. Lockyer had just related. The further history of the case was of great interest, as in February, 1909, she came again with similar symptoms pointing to trouble on the left side. The abdomen was reopened, more free blood was found in the pelvic cavity, and this time the left ovary showed a rent with attached blood-clot. Dr. Lockyer's report on the case was as follows: "This specimen reveals the presence of very degenerate chorionic villi in the free blood-clot. This was attached to the top of a hæmorrhagic area on the upper pole of the ovary. This area has been examined. It shows a corpus luteum distended with clot and with very degenerate lutein cells as a lining; in this clot there are no villi, but it is quite probable that the ovum was once inside it. The section prepared from the tube reveals the presence of chronic salpingitis. The plicæ are thickened by a deposit of fibrous tissue, and there are some recent peritonitis and injected vessels under the peritoneal coat." From the nature of the condition at the time of the operation this certainly appeared, from the operator's point of view, to be a case of ovarian pregnancy, but the parts that were removed were such

as would probably fail to convince an independent audience that the pregnancy was ovarian, and, after talking the matter over with Dr. Lockyer, they agreed not to report it as a case of ovarian pregnancy; but the history of the case left one in considerable doubt as to whether it might not have been in reality a double ovarian pregnancy, or whether it was a case of repeated hæmatocele due to a ruptured lutein hæmatoma, or whether it was a lutein hæmatoma in the first instance and an ovarian pregnancy in the second.

Dr. BLACKER said that he thought he had met with a case of this kind some years ago. The patient, a young girl aged 19, a virgin, was admitted into University College Hospital with a history of acute pain in the abdomen. The pain had commenced suddenly two days before the period was due, and was followed by the formation of an abdominal swelling which gradually increased in size. The periods had been regular, and the last period occurred at the proper date and was normal in amount. Dr. Herbert Spencer opened the abdomen and found a large hæmatocele, which was evacuated and the cavity drained. The patient made a good recovery. There was nothing to suggest pregnancy, and there seemed no reason to doubt that this was a case of hæmorrhage from a ruptured Graafian follicle in the ovary.

Obstetrical and Gynæcological Section.

February 10, 1910.

Dr. H. MACNAUGHTON-JONES, President of the Section, in the Chair.

Modern Methods of Delivery in Contracted Pelves.

By E. HASTINGS TWEEDY, F.R.C.P.I.

THE changes recently introduced into operative obstetrics have become sufficiently well established to enable us now to lay down definite rules for precise treatment in any given case of contracted pelvis. In order to do this an accurate knowledge of the pelvic measurements is necessary. Such knowledge can be obtained by the use of Skutsch's pelvimeter, and in no other way, so far as I know. It is certain that a knowledge of the length of the diagonal conjugate by no means gives us an accurate indication of the length of the true conjugate. From a theoretical standpoint there is nothing that can be said in favour of such a method, and to those who have made themselves practically efficient in the use of Skutsch's pelvimeter, the hopelessness of trying to deduce any accurate data from an estimation of the diagonal conjugate is only too well known.

Like all other arts, pelvimetry has to be learned. The proper use of Skutsch's pelvimeter necessitates considerable practice. In the hands of an expert its records are accurate to within $\frac{1}{8}$ in. Practically this is sufficient for the adoption of a correct line of treatment. Theoretically the finding of the pelvic measurements will furnish us with only half the required data, for the size of the foetal head will still be unknown; but practically this unknown quantity may be disregarded. It is found that the children born at full term of mothers with contracted pelves are up to, or even beyond, the average weight. In my last sixty-one cases of

contracted pelvis with a true conjugate of $3\frac{2}{3}$ in. or under the children averaged $7\frac{1}{2}$ lb. weight. The size of the children may be accounted for by the fact that the presenting part is unable to sink into the pelvis; therefore the cervical ganglia are not pressed on and escape the stimulation capable of starting the processes of labour at term, consequently these women do not fall into labour until after full term. Whether this be the true explanation or not, the fact remains that it is unsafe to assume that a full-sized foetus is not carried by the deformed woman.

There is a generally accepted belief that the relative proportion of head and pelvis can be ascertained by Müller's manœuvre—i.e., pressing the head into the pelvis under deep anæsthesia. As a matter of fact, this plan often ends in disappointment, for, unless the head lies in flexion, the method fails in its purpose. It will also fail if the foetus lies in the third or fourth position, and in obese women it becomes a matter of considerable difficulty to carry out the manœuvre. At best it is but an adjunct to treatment based on pelvic measurements; by itself it is far inferior to the latter.

It is convenient to classify pelvic contractions into five degrees, the first four of which are separated from each other by $\frac{1}{2}$ in. measurements in the conjugate diameter, whilst the fifth comprises all pelves with a true conjugate measuring less than $2\frac{1}{4}$ in. This classification is based on the assumption that a simple flattened pelvis has been measured. The treatment for generally contracted pelvis of a certain degree is that of a simply flattened pelvis with a true conjugate $\frac{1}{2}$ in. shorter. When the degree of deformity is known, treatment becomes simple and certain.

Contractions of the first, and even of the second, degree are seldom diagnosed with certainty unless complications arise suggesting the advisability of taking the pelvic measurements. They may lead to abnormalities of presentation, prolapse of the cord, premature rupture of the membranes and prolonged first and second stages. In the absence of such complications, and even in spite of some of them, a woman may be expected to deliver herself safely if given ample time for the several processes, and provided the child is not abnormally large. In such cases trouble usually arises from meddlesome interference, attempts to deliver when the rim of the os is still felt or the head not fixed in the brim by its largest diameter. The employment of Walcher's position to assist fixation of the head in the brim, and the application of forceps when the head has properly fixed, are both aids that may hasten successful delivery. It is only in the event of danger to mother or child arising that operations for enlarging the pelvic brim need be considered.

Induction of premature labour is never required, nor is prophylactic turning justifiable when employed as a measure to aid delivery.

The question of treatment becomes much more difficult in the third degree of contraction with a conjugate measuring between $3\frac{1}{4}$ in. and $2\frac{3}{4}$ in. With such measurements normal delivery is neither to be looked for nor expected. If the patient is healthy and in suitable surroundings, and is seen just before labour starts or early in the first stage, the classical Cæsarean section offers a satisfactory means of delivery. When performed under favourable circumstances it is almost a perfect operation. If the woman is long in labour, with the membranes ruptured, symphysiotomy or pubiotomy should be preferred, whilst perforation is the obvious resort if the child is dead.

In the fourth degree, a pelvis with a true conjugate between $2\frac{3}{4}$ in. and $2\frac{1}{4}$ in., the contraction is too great for pubiotomy or symphysiotomy. If the child is alive, Cæsarean section, intra- or extra-peritoneal, offers the only means for rational delivery. If the child is dead, perforation with cephalotripsy and cleidotomy can be relied upon for delivery. When the true conjugate measures less than $2\frac{1}{4}$ in. abdominal section is the only method of delivery to be considered. Mutilating operations in these cases are very difficult and highly dangerous, therefore not to be recommended. I have had an opportunity of repeating Cæsarean section on three of our patients. In all these there was no visible stretching of the abdominal scar; there were no adhesions between the intestines and uterus or between the uterus and abdominal wall. The fine silk (No. 3), which was used exclusively to close both the uterus and abdominal fascia, had been completely absorbed, although in one case only eleven months had elapsed since the previous operation. Still more satisfactory is the fact that the uterine scars could not be seen and were felt with difficulty.

In spite of these successes I have only felt justified in performing classical Cæsarean section thirteen times during the last six years. Many of my cases were not seen sufficiently early, or else had conditions present which contra-indicated the operation. That this is the experience of others whose skill in the performance of the operation places them beyond criticism is evidenced by the opinions expressed at the recent meeting of the British Medical Association. It was there declared that craniotomy on the living child was justifiable, and to be recommended if conditions did not seem favourable to the success of Cæsarean section. Personally I do not subscribe to such an opinion, and have never found it necessary to perforate a living child. I have to

record one fatality in these thirteen operations, the woman collapsed whilst under anæsthesia, immediately after the removal of a very adherent placenta. The operation was attended with singularly little hæmorrhage, and death was attributed to shock—a condition not uncommonly seen in association with adherent placenta.

Much has recently been heard of the operations of symphysiotomy, pubiotomy, and hysterotomy, and many think that these are simply rivals to classical Cæsarean section. Were this so, I should not be concerned in recommending them to your consideration. They neither compete with Cæsarean section nor even with each other; they are complementary operations, each with its own field of usefulness. Until this is clearly realized it will be impossible to stay the present sacrifice of life which has resulted, and must result, from such obsolete procedures as induction of premature labour, prophylactic turning, high forceps, and perforation. It is only a matter of a few years before all these methods of delivery will be viewed with the utmost abhorrence. Not only will it be considered criminal to perforate the head of a living child, but to permit one to die because of delay in delivery or from obsolete methods will rightly be condemned. Symphysiotomy or pubiotomy is to be highly recommended when danger symptoms to mother or child arise during labour with a pelvis not smaller than $2\frac{3}{4}$ in. in the true conjugate. With both delivery can be easily accomplished, with both convalescence should be uneventful, simple and painless, and with both the patient may be expected to sit up in bed on the fourteenth day, in a chair on the fifteenth day, stand and take a few steps on the sixteenth day, and walk freely from the seventeenth day. Union in both should be complete, and subsequent pregnancy, if not made easier, should be certainly not more difficult. In favour of symphysiotomy is the fact that no special instruments are required and hæmorrhage is not severe, whilst pubiotomy gains by reason of its greater simplicity and diminished liability to injury of the bladder. Particularly is this so when the subcutaneous method of Professor Bumm is employed. My personal experience of these operations is covered by eight cases—two symphysiotomies, five pubiotomies after the manner described by Professor Döderlein, and one subcutaneous pubiotomy. It is of this latter that I particularly want to speak, for in it I feel that we at last possess a plan so simple, and so safe in technique, that we are justified in recommending it for adoption by the general practitioner as a means of successful delivery in obstructed labour.

THE OPERATION.

The patient is placed in the cross-bed position, shaved, and disinfected. The vagina is tightly plugged with pledgets of sterile cotton wool wrung out in weak lysol solution. This is an important step in the operation. The rugæ of the vaginal wall are smoothed out, making the action of the antiseptic more efficient. But its most useful action is dilatation of the vagina, which permits ready and rapid delivery without fear of extensive tears of the soft parts. This plug remains in place about half an hour whilst the preparations for the operation are finished and the anæsthetic is given.

The operator, seated between the patient's legs, which are allowed to hang down, removes the plug, douches the vagina, and empties the bladder. The site of puncture should be on the skin outside the labium majus. Without any preliminary incision the needle is plunged down to the under-surface of the pubes. A finger in the vagina guides its further progress. The needle is made to hug the back of the pubes until it emerges on the abdomen just above the pubes and $\frac{1}{2}$ in. to $\frac{3}{4}$ in. to the side of the symphysis. A Gigli saw is hooked on to the needle and pulled through the wound when the needle is withdrawn. The bone is now divided in the usual manner, without any laceration of the soft parts. This is surprising, for without experience of the operation one would be inclined to believe that laceration would be severe. The fact is that the soft tissues cling so tightly to the saw that they move with it as the bone is being severed. A finger placed on the front of the pubes gives immediate warning of the severance of the bone. An assistant at either side of the patient prevents the pelvis springing apart too suddenly. Labour may be terminated immediately by forceps or version, or the patient may be allowed to deliver herself naturally, provided the child's life is not in danger. The hæmorrhage which spouts from the punctures when the saw is removed is easily controlled by digital pressure. After the third stage is completed a compress of gauze is the only dressing required. The binder is applied as usual, and, to give greater support, a broad canvas belt is placed on the outside of the binder around the trochanters. A navy's belt answers the purpose admirably.

The patient is turned from side to side in twelve hours and can turn herself on the third day. On this day the bowels should be moved; for this the patient is elevated on to the bed-pan by the binder. This raising causes no pain, and the patient is always lifted in this manner when the draw-sheet is renewed. When the binder is to be changed

it is slipped into the hollow of the back, the patient is raised by the belt, and a new binder laid under her. The belt is then slipped up and taken out, the patient raised by the binder, and the belt replaced outside the binder as before. The wound is completely healed within two or three days, and it is impossible to find the puncture-marks after a short time.

In performing Döderlein's pubiotomy there is greater fear of infection, as a compound fracture is produced; therefore asepsis should be thorough and complete here, as in all obstetrical operations. The incision over the pubes should be vertical, not horizontal. In this way the ligaments are severed and free access to the bone obtained. The bladder should be separated by pressing a gloved finger through this wound. The needle should be made to hug the bone as closely as possible and to emerge outside the labium majus. In both these operations more than one saw should be provided, as they are liable to break.

The technique of symphysiotomy is too well known to need description. There are only a few points to which I desire to call attention. The incision, not longer than 1 in., should be made on the abdomen above the pubes down to the symphysis. This incision completely obviates the danger of hæmorrhage or injury to the urethra. The gloved finger is pushed into this wound and behind the pubes, separating the bladder. With the finger in the wound, the cartilage is cut from above downwards and within outwards. The finger thus protects the bladder from injury. The sub-pubic ligament is not severed if delivery can be accomplished without it. No drainage is necessary. Subsequent treatment and convalescence are exactly the same as in Döderlein's pubiotomy, and the results are equally good.

Were these the only operations at our disposal the treatment of contracted pelvis would still be imperfect and unsatisfactory. There would be no provision for the treatment of the higher degrees of contraction when the patient is far advanced in labour; for such cases forceps and turning are quite impracticable. Perforation is not permissible if the child is alive. Symphysiotomy and pubiotomy should not be performed if the true conjugate is less than $2\frac{3}{4}$ in. When these cases are advanced in labour, with ruptured membranes, with the uterus retracted on the fœtus, with Bandl's ring well up towards the umbilicus, indicating a marked expansion of the lower uterine segment, they are notoriously unsuitable for classical Cæsarean section. It is precisely for

these conditions that extra-peritoneal Cæsarean section or hysterotomy is indicated.

At present there is an erroneous view that this operation should not be performed if there is a suspicion of the presence of sepsis. Provided the child is alive, the possibility of sepsis should not prevent the operation, for neither the general peritoneal cavity nor the cellular tissue is exposed to any extent, and no unclosed spaces are left when the operation is finished. The risk of sepsis is not increased. To my mind, the non-exposure of the peritoneal cavity and cellular tissue is the greatest advantage it possesses. Therefore, I believe that the true extra-peritoneal operation developed by Sellheim is based on wrong principles, for in it the cellular tissue is extensively exposed.

In the operation I am about to describe the intestines need never be seen. Escaping liquor amnii or blood cannot gain access to the general peritoneal cavity. There is no scar to which intestines might adhere. There is no danger of rupture of the uterus in a subsequent pregnancy. Ample room is provided for easy and rapid delivery, whilst the incision passes through a thin wall of relatively non-vascular tissue. Finally, the more advanced the labour and the more marked the lower uterine segment, the simpler becomes the operation.

THE OPERATION.

With the usual aseptic precautions, a transverse incision is made from one anterior superior spine to the other, passing about 1 in. above the pubes. This incision is carried down to, and then through, the sheath of the rectus. The upper edge of the wound in the rectal sheath is separated upwards from the muscles, and the latter are divided with the handle of the knife. The transversalis fascia is then broken through and the peritoneum incised transversely. The bladder is raised, putting the vesico-uterine fold of peritoneum on the stretch. A small opening into this fold permits the passage of a finger between the uterus and bladder. On this finger the peritoneum is divided across the whole width of the vesico-uterine fold. The upper edge of the incised peritoneum is seized and separated upwards from the uterus. Its edge is then stitched to the upper edge of the transverse incision in the parietal peritoneum, to shut off the general peritoneal cavity from the field of operation, and to inclose the intestines in a bag of peritoneum. Interrupted sutures should be used, as a continuous suture causes puckering and contraction of the opening. The sutures should be placed well

out to the lateral aspect of the opening, closing the angles. If this is not done communication is not shut off and fluids may enter the abdominal cavity, or the intestines may appear at the angle of the wound. It is not necessary to stitch together the two layers of peritoneum above the bladder.

The exposure of the lower uterine segment thus obtained permits easy delivery. The lower uterine segment and cervix are opened longitudinally. If the head presents, it is extracted with the left blade of the forceps and one hand. If any other portion of the child presents, it is extracted manually. The cord is clamped and cut. Time is given to allow partial separation of the placenta. During this pause a hypodermic injection of $\frac{1}{50}$ gr. of ergotin in may be administered. Post-partum hæmorrhage was a complication in my three cases. The placenta and membranes are removed, the uterus douched and then plugged with iodoform gauze, the end of which is pushed through the cervix into the vagina. The uterine incision is closed by interrupted catgut sutures; the free edge of the peritoneum immediately above the bladder is united by interrupted catgut sutures to the upper portion of the peritoneum from which it was originally severed. The sheath of the rectus is closed with continuous No. 3 silk sutures, and the edges of the skin-wound brought together by Michel's clamps or in any manner convenient to the operator.

To sum up, my views on the treatment of contracted pelvis are:—

- (1) Induction of premature labour is never advisable.
- (2) Perforation is not permissible unless the child is dead.
- (3) Turning should never be employed as a treatment for contracted pelvis, but may still be performed for complications of labour, such as prolapse of the cord, when associated with contractions of the first and second degree.
- (4) In the greater degrees of contraction time should not be wasted in an endeavour to obtain natural delivery.
- (5) On the other hand; in the lesser degrees ample time should be given the woman to enable her to deliver herself, if possible. Eight or ten hours may be necessary for the moulding of the head, and interference should not be considered until there are evidences of foetal or maternal distress. Once foetal symptoms of distress are manifested there should be no delay in delivery. Walcher's position should not be forgotten as an aid to fixation of the head.

(6) High forceps should never be applied until all arrangements are perfected for an operation to enlarge the pelvis. It is, in my opinion, a pity to proceed to the latter expedient until forceps have been tried tentatively. It must be confessed they occasionally accomplish their purpose under the most unexpected circumstances.

Finally, in these as in all other obstetrical operations, the best results cannot possibly be obtained if rubber gloves are not worn.

DISCUSSION.

Dr. CHAMPNEYS said that the Section was much indebted to Dr. Hastings Tweedy for his very clear and interesting paper. His opportunities as Master of the Rotunda Hospital were great, and for the working out of new problems such a position gave unusual advantages. There were certain propositions in the paper which were at variance with his (the speaker's) present opinions: for instance, the absolute condemnation of the induction of premature labour. This, in suitable cases, had always seemed to him to be a good and sound proceeding, and one which had, in his hands, produced good results. He published not long ago the account of a case in which he delivered a premature child through a pelvis whose conjugate was not more than $2\frac{1}{2}$ in. Not only was the child now alive, but was 17 years old, healthy and bright, and earning her own living. The mother, too, was well, and had been seen by others, so that there was no doubt about the measurements. This was an extreme case, and he did not hold it up as a precedent. Then the results of symphysiotomy and pubiotomy related by Dr. Tweedy struck him as being exceptional. He had read of all sorts of disasters—injuries to the soft parts, to the bladder, trouble with union, and a not inconsiderable mortality, both maternal and foetal. Moreover, the limits of their application were quite narrow. Up to the present time he had treated cases of contracted pelvis with induction of premature labour and Cæsarean section, and had been well satisfied with the results. He promised, however, to give the proposals of Dr. Tweedy careful consideration. He begged again to thank him for his interesting and instructive communication.

Dr. HERMAN thought the Society was to be congratulated on having the opportunity of discussing Dr. Hastings Tweedy's able and original paper. He was interested in Dr. Tweedy's commendation of Skutsch's pelvimeter. He (Dr. Herman) had tried to use Skutsch's pelvimeter, but had not had much success with it. Possibly with more perseverance he would have come to better appreciate its value. He hoped those Fellows of the Society who were actively engaged in the teaching of midwifery would note Dr. Hastings Tweedy's remarks on this subject and endeavour to make themselves, if not already

so, and their pupils, expert in the use of this valuable instrument. There were many pelvimeters with which it was easy to measure accurately the true conjugate in the dry pelvis, but when they came to apply these to the living patient they found the bladder and urethra in the way. He could not agree with Dr. Tweedy in regarding the size of the child as a factor that might be neglected. On the contrary, he thought the relative size of child and pelvis was much more important than the absolute measurements of the pelvis. Whether the child weighed 7 lb. or 10 lb. was as important as the length of the conjugate. He found it generally easy to judge of the relative size of head and pelvis by pressing the head down into the pelvis with the hands on the abdomen, although he admitted it was not always so. Dr. Tweedy mentioned as a cause of difficulty the case of an obese patient. That was so, but women during the child-bearing years were not often obese. Dr. Hastings Tweedy would have us abandon the induction of premature labour, prophylactic version, the high forceps operation, and craniotomy while the child was living. Acceptance of Dr. Hastings Tweedy's views involved acceptance of one of two propositions—either (1) that the mortality of Cesarean section was not greater than that of delivery through the natural passage; or (2) that the life of the child was to be considered before that of the mother. As to (1), the most recent collection of cases that he knew of, that of Dr. Munro Kerr, showed a mortality of about 8 per cent. As to (2), he (Dr. Herman) adhered to the old principle that the life of the mother was always to be preferred to that of the child. Dr. Hastings Tweedy put side by side the two operations for widening the pelvis—symphysiotomy and pubiotomy—as about equal in difficulty. It was evident from this that he was not acquainted with subcutaneous symphysiotomy. He spoke of an incision an inch long, and of putting the finger down behind the symphysis. In the subcutaneous method of performing symphysiotomy the only instrument needed was a small tenotomy knife, the only incision was a mere puncture, and the duration of the operation was a few seconds. He could not imagine any one acquainted with subcutaneous symphysiotomy preferring pubiotomy. The real objection to methods of widening the pelvis was that for their safe use it was necessary that the relative size of the head and pelvis should be determined with great accuracy, for dragging too large a head through the widened pelvis led to disaster. Lastly, on one point he differed not only from Dr. Hastings Tweedy, but from most recent obstetrical writers. He did not think, as Dr. Tweedy did, that indiarubber gloves were necessary for the attainment of the best results. Puerperal fever had been banished from the General Lying-in Hospital without the use of indiarubber gloves. Lord Lister had obtained the splendid results which convinced the world of the value of antiseptics without gloves. He (Dr. Herman) had asked persons whose occupation was such that they saw a great deal of operating and its results whether there was any difference between the results of those surgeons who wore indiarubber gloves and those who did not; and the reply always was that they perceived no difference. In large hospitals and in West-end private practice the cost of indiarubber gloves was a trifle

compared with other things. But in the modest budget of a poor midwife the cost of indiarubber gloves was a serious matter. He thought indiarubber gloves were like the carbolic spray—a fashion which would pass away.

Dr. HERBERT SPENCER said the Section was much indebted to the Master of the Rotunda for his valuable and lucid paper. With much of the paper, which was based upon the large experience of Dr. Tweedy, he found himself in agreement. On the question of induction of premature labour he disagreed absolutely, and he did not think any evidence had been brought forward in favour of pubiotomy as a substitute for induction, whatever might be said for it as an emergency operation during labour at term. He was more in agreement with the views expressed by Dr. Herman and Dr. Champneys. He could not allow that induction of premature labour was an obsolete operation, or one which should be viewed with abhorrence. He had been practising induction for twenty-three years, and was more in favour of the operation than ever. He did not remember a single case in which any serious trouble occurred to the mother, who recovered quite as well as, if not better than, after a normal labour at term. The proportion of still-births was small, and, although of course a considerable number of the children did not grow up, that was largely due to want of care or absence of milk in the mothers rather than to the premature delivery. One patient on whom he had induced premature labour for contracted pelvis on six occasions had five living healthy children, who often came to see him at the hospital. She would probably have six living had not the assistant unnecessarily, and against the speaker's rule, applied forceps to deliver the head. Two days ago he had seen a lady on whom he intended to induce labour shortly for contracted pelvis with a $3\frac{1}{2}$ in. conjugate. He had done so in the only two previous pregnancies; the mother was in perfect health, and the two children were as healthy and as strong as any he had seen. Seventeen years ago he had induced labour in a woman with a 3 in. conjugate, and the child had grown up a strong, healthy girl. There was no doubt about the size of the conjugate, for the pelvis was in his possession, the patient having recently succumbed to an operation. In view of such cases, he held strongly the opinion that induction of premature labour was an admirable operation, of a simplicity which enabled it to be carried out successfully by any doctor, practically free from risk to the mothers, and giving fairly good results in the case of the children. In order to be successful, however, it needed to be done in the proper cases, at the proper time, and in the proper way. In his opinion the proper cases were those in which the pelvis measured not less than 3 in. in the conjugate, the proper time the thirty-fifth week or later, the proper way the bougie method. Champetier de Ribes's bag, which Dr. Herman and the speaker had first recommended in this country many years ago, was inferior to bougies for this purpose. With regard to Dr. Tweedy's advocacy of pubiotomy, he might say that he (the speaker) had never performed either symphysiotomy or pubiotomy, preferring induction of premature labour and Cæsarean section in the interest of the mother in clean cases. In really infected cases any operation

(pubiotomy included) was attended with great risk to the mother. No one would deny that more children survive after pubiotomy than after induction of premature labour, but the important point was whether the superiority of pubiotomy in this respect was sufficient to justify the increased risk and disadvantages to the mother. He agreed with Dr. Herman that the interest of the mother should come first; in the interest of the child, and, in many cases at least, in the interest of the mother, too, Cæsarean section was better than pubiotomy. Dr. Tweedy's remarks were the outcome of a personal experience of two cases of symphysiotomy and six cases of pubiotomy, of which only one was done by the subcutaneous method he recommended. He said nothing of any ill-effects on the mother, though records of such ill-effects exist in abundance. In Schläfli's¹ investigation of 510 cases of pubiotomy, hæmatoma was observed in 17 per cent., severe tears in 15·4 per cent. (of these 12·5 per cent. died), simple tear of the soft parts in 18 per cent., injuries to the bladder in 12 per cent., thrombo-phlebitis in 8 per cent. Of 120 cases investigated later, hernia through the gap in the bone was found in 7·5 per cent., prolapse of the vagina in 24 per cent., chronic incontinence of urine in 4 per cent. Of the mothers, 4·9 per cent. died; of the children, 9·6 per cent. With these results v. Herff compared his own statistics of induction of premature labour—viz., in 117 cases one maternal death occurred, and 80 per cent. of the children left the clinic alive. The results of fifty-three cases of pubiotomy in Bumm's clinic were given by Kroemer²: in over 3 per cent. the bleeding was profuse; in five cases hæmatoma, œdema, and thrombi occurred. In twelve spontaneous deliveries there were three unimportant injuries of the soft parts—the bladder was injured three times by the needle, and healed spontaneously. Of the remaining forty-one women delivered artificially, there were seven injuries of the bladder or urethra—i.e., in nearly 19 per cent. of the cases the bladder or urethra was injured. In nineteen cases the soft parts were extensively torn by the bones. Only one mother died, but 54 per cent. had fever in the puerperium; 86·6 per cent. of the children survived. Moderate complaints ("mässige Beschwerden") were made by half the patients subsequently. In five women there was weakness in retaining the urine, and in two cases incontinence remained after cure of the fistula. With results such as these, obtained in Bumm's clinic, he did not think Dr. Tweedy was justified, with a personal experience of one case, in recommending Bumm's subcutaneous pubiotomy as a safe and simple method of delivery for the general practitioner. With regard to Dr. Tweedy's method of performing "extra-peritoneal" Cæsarean section (he understood that the peritoneum was opened), he asked Dr. Tweedy how long he took to deliver the child by his method. Personally he thought that rapid delivery of the child was of great importance in Cæsarean section to prevent asphyxia and its consequences, and he had repeatedly delivered the child through the classical incision in one minute or one minute and a quarter from the beginning of the operation.

¹ v. Herff, *Münch. med. Wochenschr.*, 1908, lv, p. 2595.

² Kroemer, *Berlin. klin. Wochenschr.*, 1908, xlv, p. 1044.

Dr. EDEN said that he would tell the Section something of the experience they had had at Queen Charlotte's Hospital with induction of premature labour for pelvic contraction. From figures taken from the annual reports, he found that during the four years 1905-8 inclusive, 309 cases of pelvic contraction had been delivered in the hospital. Of these, 101 cases, or roughly one-third, had been dealt with by induction of premature labour. Every patient entered for admission to the hospital was examined by the junior resident medical officer, and, if he detected pelvic contraction, the case was seen by a member of the visiting staff on duty. If the latter decided upon induction, the patient was subsequently seen at intervals, and the time for inducing labour was determined by Müller's manœuvre. Personally, Dr. Eden thought that the method of Müller fulfilled all the requirements of these cases. Dr. Tweedy's chief objection to Müller's method was that with a flat pelvis the head usually entered the brim extended and that in this attitude it could not be pushed down into the brim from above by the operator. But it must be recollected that this extension was produced by the engagement of the head in the brim during labour, and he (Dr. Eden) knew of no evidence that the extended attitude was common before labour had begun. The method of induction used at Queen Charlotte's was Krause's method, and the foetal mortality of the 101 cases referred to was 13 per cent.; in this were included all infants who died before the discharge of the mother from the hospital. In five-sixths of the cases the induction was performed at, or later than, the thirty-sixth week, and the average weight of the infants at birth was 5 lb. to 5½ lb. They were not allowed to leave the hospital unless their general condition was good and they were gaining weight. There was no maternal mortality. Of the cases of pelvic contraction which for one reason or another went to term, 42 were delivered spontaneously with a foetal mortality of 4·7 per cent.; there was no doubt that this termination is the most favourable for the child. Seventy-four cases in which the conjugata vera was 3½ in. or more were delivered by forceps at term. It would be agreed that such cases were suitable for this method of delivery. The foetal mortality was 18·7 per cent., and there was no maternal mortality. During the same period 35 cases of pelvic contraction were treated by Cæsarean section, the operation having been performed in the great majority of instances before labour. The foetal mortality was 11·4 per cent., but two of the mothers died, so that the operation was attended by a maternal mortality of 5·7 per cent. It would be admitted that if this operation was performed after the mother had been for several hours in the second stage of labour, as contemplated by Dr. Tweedy, the mortality would probably be much higher than this. The operations of symphysiotomy and pubiotomy had not been performed at all during the period under consideration. The results of induction obtained at Queen Charlotte's compared so favourably with the alternative procedures of delivery that he did not feel at all inclined to abandon the operation. And another consideration should be borne in mind. The practice of midwifery was not confined to lying-in-hospitals; the great bulk of women were confined in their own homes under the supervision of medical

men who had not had the same opportunities of acquiring special experience and skill as the members of that Section. It was therefore very desirable for obstetric teachers to retain the simpler procedures as long as they yielded satisfactory results, and to teach and demonstrate them for the benefit of their students.

Dr. GRIFFITH said that Dr. Hastings Tweedy in his paper condemned in a very definite manner, as many continental obstetricians had in recent years, the British adherence to the induction of labour in preference to the routine operation of symphysiotomy and pubiotomy which for many years have been extensively practised on the Continent, and but rarely in England. He personally was satisfied that each operation had its own sphere of usefulness and was not to be condemned, but he was not prepared to accept the extreme teaching of some obstetricians, that in all cases where the foetal head would not descend through the brim symphysiotomy or pubiotomy was necessarily the best, and indeed the only good, method of treatment, when the contraction of the conjugate was not under 3 in. The criterion of success was to deliver an uninjured child capable of healthy development with the least injury to the mother. For many years he had demonstrated and practised a method for determining the probability of a child passing through the pelvis which he believed to be superior to Müller's. Having ascertained the head was presenting, or by external version had made it present, he placed the patient in an arm-chair, in such a position that her uterus was vertical, for twenty minutes. The difference in weight of the foetus (a few ounces) and the liquor amnii was sufficient to cause the head to descend into the brim. So long as this happened induction was unnecessary, but when a period arrived when the head could no longer in this way enter the brim, induction should be performed, and he had found it generally successful. This method determined the date of induction, without any necessary reference to the exact length of the conjugate, and it was common experience that the length of the conjugate, except in extreme cases, did not determine, as Dr. Tweedy had pointed out, the difficulty of delivery. He had not unfrequently by this method been able to allow pregnancy to go to full time in cases in which induction had been performed on previous occasions.

The PRESIDENT (Dr. Macnaughton-Jones) said that it required a man of considerable experience in obstetric work to venture to intrude any observations into such a debate as this, and he should not do so were it not that so many points of extreme importance were raised by the communication of Dr. Hastings Tweedy, in which he took a great interest. For some eighteen years he was very largely engaged in the practice of midwifery in public institutions and in private practice. This involved the fulfilment of public duties, either as a temporary substitute or permanent officer in eight of the nine Poor Law Dispensary Districts of Cork, with its 80,000 inhabitants, apart from his private practice. Initiating the present Cork Maternity in 1872, he had in it ten years of obstetric work. The only statistics that he had now before him were the

first five years of the Maternity; and he was willing to acknowledge that he was exceptionally fortunate, for during the entire period of those eighteen years he had only two maternal deaths in difficult labour from pelvic deformity, and these occurred from sepsis after prolonged and neglected labour, before assistance was sought for. He (the President) thought that we should not run away with the idea that older methods gave us altogether such bad results. Of the 1,611 cases attended by him and his colleagues during the period he mentioned, 14 mothers died, and 64 children. There were 93 forceps cases, and 3 craniotomies, 3 eviscerations, 2 Cæsarean sections, and 17 versions; a mortality of 0·8 per cent. of mothers, and 3·9 per cent. of children. Taking all the other cases, preternatural and complex, into consideration, and remembering that the majority of the women were attended in the worst of surroundings in the poorest slums of the city, when epidemics of smallpox, typhus fever, and scarlet fever were prevalent, and assistance often not sought for until far on in the labour, this did not, he thought, compare unfavourably with a similar number of cases attended under more promising conditions at the present time. For we must always recognize that classical midwifery in hospital and among the rich is one matter, and that which is the outcome of emergency and in the homes of the poor a very different thing. The rules we lay down dogmatically in the one must be materially modified in the other, when we have to consider the elements contributory to mortality in the absence of the essentials of aseptic technique, proper appliances, and the previous presence of septic conditions. In those days the great gain of the *Walchian* position in delivery by forceps was not recognized. Modern obstetrics teach us some things clearly:—

(1) That one cannot separate the character of a labour from the state of a woman's health and physical state during pregnancy.

(2) That whenever it can be secured, a careful examination of the pelvis should be made, and an estimation of its capacity and anatomical features should be arrived at; and if there be any doubt as to the relative proportions of foetal head and diameters, this examination should be periodically repeated, especially towards the end of term. And this examination, whenever it can be availed of, should be supplemented by radiography, carried out by the double spectroscopic method, before the woman is subjected to the test of labour; it being borne in mind that the Röntgen ray is liable to cause abortion in the early months of pregnancy.

(3) That prolongation of pregnancy beyond the normal term is attended with greater risk to mother and child; a risk considerably increased if there be any abnormality of the pelvis present, and consequently no woman should be allowed to go over time if there be any degree of contraction present.

From McKerron's researches it would appear to have a bearing on the size generally of the foetus, the circumference and ossification of the foetal head, and morbid placental changes; while there also is, on the maternal side, loss in the abdominal power. It seemed to him to be essential that, in order to arrive at any satisfactory conclusion as to the cranial and pelvic relationships in such cases, an anæsthetic is necessary, the patient being first properly

prepared and the Müller bimanual procedure adopted. He wished also to express his strong feeling in favour of the induction of labour within prescribed limits of deformity carefully carried out with the necessary preliminary precautions, at any time after the thirty-sixth week, according to the exigencies of the case, so as to save the mother the risks of operative procedure and give the child a fair chance. Nor did he think we should altogether discard, in a certain minority of cases, version as an alternative to the forceps, where the degree of contraction is not great, the presentation posterior parietal, and the turning carried out early, when the os is fully dilated and the membranes intact. There is no more dangerous interference than that of the high forceps delivery in the case of a cranium that cannot engage the brim. The day of perforation of a living child's head, he considered, had passed. In face of the obvious advantages from induction of labour, and the modern operative steps, this may be accepted as an axiom.

Arising out of the discussion, he should like to express his personal feeling that he doubted if any method of pelvimetry for use in general practice superseded that of the hand and finger, carried out in the proper manner. He was pleased to see that this was the opinion expressed by Professor Jardine in his classical work on clinical obstetrics just issued from the press. It can be well assisted by Müller's bimanual method of testing the engagement of the brim, or that which Dr. Monro Kerr has described, and which is habitually carried out in the Glasgow Maternity. Though it may sometimes happen that a labour is completed naturally or by forceps which would appear to demand Cæsarean section, in the face of the evidence of the favourable results from either pubiotomy, symphysiotomy, or Cæsarean section, it would seem to be of supreme importance to differentiate before the last month of pregnancy, or at the earliest possible time of the labour, the case that absolutely demands operation, and which step is preferable so as to avoid many of the risks that follow after labour has progressed for any time. There will ever be a class of case distinctly within the scope of surgical interference on the one hand, or which we can without fear entrust to nature and the ordinary obstetrical aids on the other. These cases must be left to the individual judgment of the obstetrician, and his personal skill and aptitude in the conduct of such a case, whether operative or otherwise.

He desired to convey on behalf of the Section, to Dr. Hastings Tweedy, their warmest thanks for a paper which had led to so interesting and valuable a discussion; and though in some points the speakers had differed from him, still it was by calling forth such expressions of opinion, and eliciting different views, that correct conclusions were arrived at and progress made.

Dr. HASTINGS TWEEDY, in reply, said he could not but contrast the thoughtful attitude of Dr. Champneys with Dr. Herman's uncompromising hostility in regard to the subjects under discussion. Dr. Herman's adverse criticism extended even to the use of rubber gloves, which he characterized as a passing fad. His words would have more weight if he could produce authority,

personal or otherwise, for saying that they interfered with manipulative skill after having been continually used for a period extending over a few months. Dr. Herbert Spencer cited some isolated cases in which brilliant results had followed the induction of premature labour. Until accurate dates of the period of induction were available, and also the sizes of the children born, there could not be too much importance attached to these deliveries. The statistics he quoted from Professor Bumm's clinic cannot be cited as a criterion for the modern operations performed under the strict conditions indicated in Dr. Tweedy's paper, for the complications enumerated are in nearly every case preventable by a more exact technique and careful selection of cases. Professor Bumm's statistics include all the bad results obtained by himself and his assistants during the development of the operation, when experience was not sufficient to enable the operator to avoid the complications mentioned. Dr. Eden's statistics of induction of labour at Queen Charlotte's hospital, with a foetal mortality of 13 per cent., are very much better than those obtained in other clinics, where the methods of induction do not differ from those employed by Dr. Eden. An explanation for this discrepancy will be found in the date at which labour is induced. If he waits until the thirty-fifth week or later, it is obvious that he is inducing labour in cases which would fall under the first or second degree of contraction, which in the Rotunda Hospital would be left to nature with a smaller resultant mortality than 13 per cent. In reply to Dr. Stevens, when foetal death occurs it is due either to faulty technique or to the selection of a wrong operation in a given case.

Fibroid of the Vaginal Wall.

By HENRY RUSSELL ANDREWS, M.D.

R. C., AGED 47, had had thirteen pregnancies, none of which had gone further than six and a half months. The last pregnancy was seven years ago. Three years ago she noticed a lump protruding from the vagina. A plastic operation on the anterior vaginal wall was performed at a provincial hospital, but the stitches gave way during a fit of sneezing and the condition was in no way improved, and incontinence of urine was added to her discomfort. Later she was operated on in London by a general surgeon, who performed the operation of ventral fixation of the bladder. In spite of prolonged rest after this operation the swelling, which bulged into the vulva, persisted. The patient also complained of frequency of micturition, the bladder being emptied every hour. Vaginal pessaries of many sorts and sizes were tried, but with no benefit. The

discomfort was so great that the patient was unable to do her household work.

On examination under an anæsthetic, a swelling was found which appeared at first to be a cystocele. More careful examination, however, showed that a firm, more or less solid tumour, about the size of a fist, hung from the anterior vaginal wall, to which it was attached by a broad pedicle. It was covered by thick œdematous mucous membrane, which showed numerous scars, some due to old ulceration and some to the plastic operation that had been performed. No connexion between the bladder and the tumour could be found. Bimanual examination was unsatisfactory, owing to the size of the tumour ; but the fundus of the uterus could be felt low down in Douglas's pouch with the cervix, looking upwards and forwards, at a much higher level. There seemed to be some obstruction to the outflow of blood from the uterus, as pushing up the fundus uteri produced a flow of thick tarry blood with rather offensive smell. An incision was made round the base of the tumour, through the vaginal mucous membrane, and the tumour was separated with ease, very little bleeding being caused. Redundant vaginal mucous membrane was removed and the wound closed by a vertical suture. The patient is now free from discomfort and there is no frequency of micturition.

The tumour was about the size of a cricket ball, elastic and almost fluctuating, and covered on the greater part of its surface by vaginal mucous membrane. On section, the cut surface was grey in colour with pearly white trabeculæ. Microscopically it is seen to be composed of fibromyomatous tissue which has undergone some myxomatous change. It was thought that fibroids of the vaginal wall were sufficiently rare to warrant the exhibition of this specimen before the Section.

A Specimen of Ectopic Gestation.

By S. JERVOIS AARONS, M.D.

THE interest in the specimen shown to-night lies in the very peculiar clinical features presented by the patient from whom it was removed, and not in the specimen itself. M. R., aged 24, single, came, accompanied by her doctor, complaining of metrorrhagia which had lasted for four weeks. Her menstrual history was as follows : The catamenia

commenced at the age of 17, and were of twenty-eight-day type, lasting from four to five days, always regular and of normal amount, and unaccompanied by pain. The last regular monthly period commenced on October 17 and ended on October 22; the November period was missed. On December 17 the flow again came on and continued up to the time of examination, January 15, and during this time had been very profuse.

On examination, the patient was exceedingly thin and exsanguine; the face was pinched and drawn, and there were heavy dark circles round the eyes. *Per vaginam*: The hymen was absent, the vagina was patulous and readily admitted two fingers, the cervix was high up and pointed forwards. Through the left and posterior fornices could be felt a firm but somewhat resilient mass occupying the left and posterior parts of the pelvis. Bimanually the body of the uterus could be felt above the brim of the pelvis and was pushed over to the right. To the left of the uterus, and closely adherent to it, was a mass about the size of a large orange. A diagnosis of ectopic gestation was made, and immediate operation advised.

On January 17 the abdomen was opened in the middle line, and the patient was then placed in the Trendelenburg position. The uterus and part of the distended left tube immediately came into view. The tube was clamped close to the uterus. The intestines were closely adherent to the tube and uterus, but were separated with little difficulty. The tube was then seen running downwards and backwards and was adherent to the posterior surface of the uterus. In the process of separating this, the tube ruptured and the mole dropped into the pouch of Douglas. The tube was then ligatured and removed, and the mole lifted out. The ovary was not removed. The right ovary and tube were normal. The abdominal incision was sutured in three layers, the dressings applied, and the patient put back to bed with a better pulse than when she was placed on the operating table. Recovery was uneventful.

It will be noticed that all the patient complained of was metrorrhagia, and that was the *sole* reason for seeking advice. For the best part of three weeks the treatment was for this symptom. When first seen on January 15, and subsequently, endeavours were made to obtain a more definite history, especially as to pain; but the most that could be elicited, after very close questioning, was that a few days before coming for advice there had been a stomach-ache which soon passed off, and which was thought to be due to some medicine which had been taken

for the bowels. On being questioned about the discharge, the reply was that "it was constant, it was bright red and profuse." No shreds were passed nor anything like a cast, and nothing of this nature has been passed since admission to the Nursing Home.

The almost total absence of the classical signs and symptoms of ectopic gestation warrant the placing of this case on record. The moral to be drawn is that no woman, married or single, who complains of metrorrhagia, should be treated without first being examined.

A Specimen of Ovarian Pregnancy.

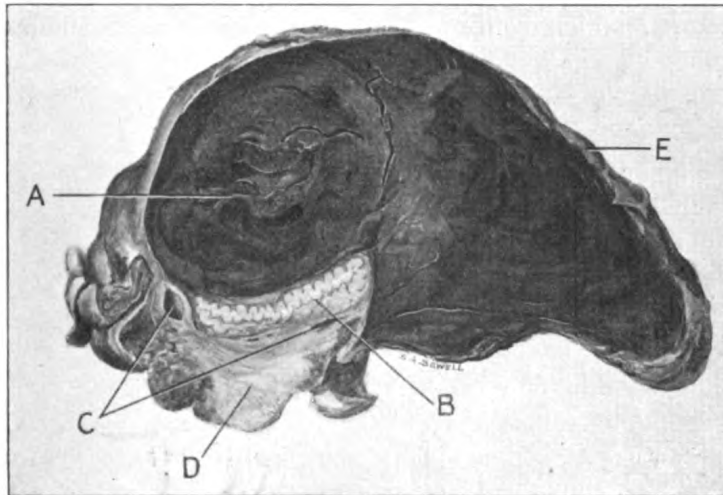
By E. HASTINGS TWEEDY, F.R.C.P.I.

THE patient, who had been married for four years, had had three children, the youngest born in June, 1909, and one abortion. She menstruated on September 18, 1909, for the first and last time since pregnancy. On November 5 she was seized with acute pain in the right lower abdomen, from which she almost fainted. Examination on this day revealed a tender mass, the size of a hen's egg, on the right side of a retroverted uterus. Diagnosis: Extra-uterine pregnancy.

The woman refused to stay in hospital, but returned on November 15 and gave a history of continuous bleeding and intermittent pain since she was last seen. On November 16 the abdomen was opened. We found both tubes and the left ovary quite normal. The right ovary was obscured by a blood-clot about 3 in. in diameter; to it was attached the ovarian ligament. The tumour was removed in its entirety, leaving the tube, which lay above it, intact. A pedicle was obtained by pulling on the posterior layer of broad ligament; the cut passed through ovarian tissue, some portion of which was left in the stump.

Report on the Specimen by Robert J. Rowlette, M.D.—The specimen, when fresh, was an elliptical mass, 3 in. in long diameter. It consists of a small mass of dense tissue, to which is adherent the larger mass of blood-clot. The blood-clot partially surrounds the piece of tissue; the surface of the clot is laminated in fibrinous strata, which easily peel off. On cutting the specimen into halves it is seen that the tissue consists of two parts: (1) A greyish-pink mass of firm connective tissue, with vessels visible to the naked eye; (2) a deep-yellow band, 1 in. long and $\frac{1}{4}$ in. wide, which is obviously what remains of a corpus luteum. This

yellow band separates the connective tissue from the clot, and it is evident that the clot had its origin in hæmorrhage into a corpus luteum, which then ruptured. The middle of the clot is softer and of deeper colour than the periphery. Microscopically sections were examined from various parts of the mass. Sections taken, including the dense tissue, the corpus luteum, and the adjacent part of the clot, show that the dense tissue is ordinary ovarian substance, covered in part with regular cellular membrane and containing many blood-vessels. The typical structure of the corpus luteum is seen; in the clot near it numerous chorionic villi are found, cut transversely and obliquely. No embryo was found. The presence of chorionic villi establishes the fact



A, blood-clot containing chorionic villi; B, corpus luteum; C, blood-vessels;
D, ovarian tissue; E, fibrin on surface of clot.

of pregnancy, and the situation of the pregnancy, in close relation to the remaining lutein tissue, shows that the pregnancy must have been situated in a corpus luteum.

Report of Pathology Committee.—The Committee have examined Dr. Tweedy's specimen and sections prepared therefrom, and are of opinion that the author's description of the specimen is a correct one.

In reply to a question from the President, Dr. TWEEDY stated that products of conception were found within the ovary.

A Specimen of Tubal Mole.

By E. W. HEY GROVES, F.R.C.S.

THE patient, M. B., aged 28, had been married three years, and had one child, aged 2. The confinement was a complicated one, and she remained in bed for seven weeks after it. Menstruation had been irregular ever since confinement, often being absent for two or three months. Her last period was two months ago. The breasts had been getting fuller, but patient had not thought herself pregnant. On the morning of January 11, on rising, she had pain and discomfort in the lower abdomen, which continued all day, and became suddenly very

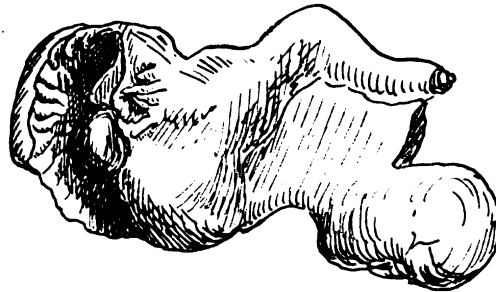


FIG. 1.

The left tube and ovary. A mass of clot is protruding from the gaping tubal ostium.

severe about six in the evening. No vomiting. She was admitted into the Cossham Hospital on January 12 in a condition of considerable shock. Her pulse was rapid (200), small, and thready; the skin pale, and the temperature subnormal. She rapidly improved after admission. On examination there were tenderness and rigidity on the left side of the abdomen below, and, bimanually, an indefinite mass was felt on the left and behind the uterus extending almost up to the umbilicus.

Operation by median incision, on January 17, in the Trendelenburg position. Several pints of dark clotted blood were removed from the pelvis. Lying loose in Douglas's pouch there was a firmly-organized clot about the size and shape of a duck's egg. This proved to be a tubal mole. The left Fallopian tube had a widely-dilated open end, from which the blood-clot was extruding. The left appendages were removed;

abdomen closed without drainage. The patient made a good recovery, and was discharged on February 6.

The specimens shown were the left appendages and the tubal mole. The proximal part of the tube was unaltered, the middle part was distended by a mass of chorionic growth, whilst the distal part was

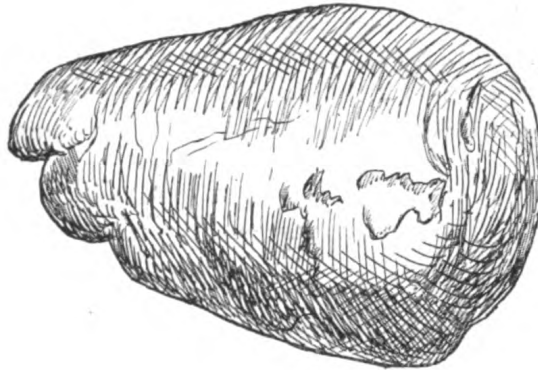


FIG. 2.

The mole before it had been opened.

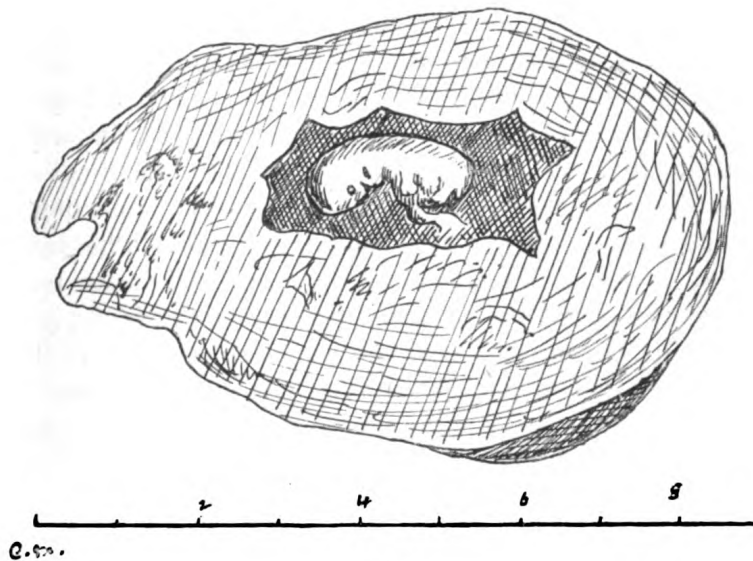


FIG. 3.

The mole after it had been cut open in its long axis. The fœtus is seen attached by a short cord to the wall of the central cavity.

widely dilated and occupied by some organized blood-clot. Clearly the mole had recently occupied the fimbriated end of this tube. The mole, which had a striking resemblance to a duck's egg, was 8.5 cm. long and 5 cm. broad. When opened it contained a smooth-walled cavity about 2½ cm. long, in which lay a foetus, 22 mm. in length, attached to the wall of the cavity by a short umbilical cord. On section, the wall of the mole was found to consist of organizing blood-clot richly permeated by a network of chorionic villi. The latter showed evidence of degeneration, inasmuch as the nuclei stained badly. The very unusual presence of a normal foetus inside a mole, whether uterine or tubal, is no doubt associated with the equally unusual preservation of the chorionic villi in the wall of the mole.

Spontaneous Transverse Rupture of the Uterus at the Fundus during Labour.

By ARTHUR H. N. LEWERS, M.D.

THE patient, from whom the specimen shown was obtained at the post-mortem examination, was a married woman, aged 38. She had had one child and afterwards three miscarriages before the last pregnancy, which ended fatally during labour. Labour began in the evening of October 26, 1909, and she was visited by a nurse belonging to the London Hospital Maternity Charity at 7.30 p.m. At that time there was a slight "show" and the cervix admitted one finger. The vertex was presenting. The pulse was regular—70. The patient was seen again at 1 a.m. on October 27—five and a half hours later. She was then much collapsed, and, from the statements of friends, it appeared she had gradually become so during the previous half-hour. The nurse at once sent up to the London Hospital for the resident accoucheur.

The resident accoucheur (Mr. R. Burgess) saw the patient at 1.30 a.m. She was then obviously very ill, her face white, lips blue, almost pulseless, very cold, sweating, restless, and with irregular respiration. The condition of the patient was quite certainly not to be accounted for by any external bleeding that had occurred. On examination, Mr. Burgess found the uterus tender and somewhat tense. The outlines of the child could not be felt well. The foetal

heart was not heard; the os admitted two fingers; the vertex was presenting, and the membranes were unruptured. There was only slight external bleeding. At this time the case was thought to be one of concealed accidental hæmorrhage. Several pints of salt solution were given *per rectum* and the patient was brought up to the London Hospital in a cab. At 3 a.m.: After admission the patient was in much the same condition. The membranes were ruptured, and normal liquor amnii escaped. The vagina and cervix were packed with gauze and a tight abdominal and perineal binder was applied. The rectal injections of salt solution were repeated. At 6 a.m.: No labour pains had occurred since admission. On examination of the abdomen, the foetus was now felt to be outside the uterus, and palpable beneath the abdominal wall very distinctly. The patient was practically pulseless. Mr. Burgess then sent for Dr. Lewers. At 7 a.m.: Dr. Lewers saw the patient at this time. She was then just alive. She showed all the signs of severe internal hæmorrhage, and the pulse at the wrist could not be felt. Intravenous injection of salt solution was performed, and at the same time Dr. Lewers opened the abdomen, the patient having been given an anæsthetic. The foetus and placenta were found free in the peritoneal cavity, which also contained a very large amount of fresh blood and clots. The uterus had contracted down to the usual size just after delivery. A large tear was seen running transversely at the fundus: it was a fairly clean tear, and seemed quite suitable for stitching. Accordingly, a series of deep sutures of silkworm gut were inserted and tied, as in the course of a Cæsarean section; but, although this took a very few minutes, the patient died as the last suture was being tied.

At the post-mortem examination it was found that the pelvis was not contracted. As regards the uterus, its cavity measured $6\frac{1}{2}$ in. from the fundus to the external os; the breadth of the cavity was 4 in. The uterus was opened by the pathologist along its left antero-lateral wall, so as to expose the cavity throughout its length. The placental site was ill-defined, but situated at the fundus and over the posterior wall. No trace of division into upper and lower uterine segments could be seen. The round ligaments were symmetrically inserted into the anterior wall at a level equidistant between the fundus and cervix. The loose attachment of the vesical peritoneum stopped at this level. Looking at the fundus from the posterior aspect, a long tear, 6 in. in length, was seen. This tear had been sutured by nine deep silkworm-gut sutures. The tear ran from the neighbourhood of the left ovarian ligament upwards towards the fundus, keeping slightly posterior to the

upper margin of the uterus. The tear passed beyond the middle line at the fundus downwards towards the right cornu, and ended $2\frac{1}{4}$ in. from the insertion of the right Fallopian tube. The peritoneum in the immediate neighbourhood of the right end of the tear was lacerated and retracted, exposing uterine muscle fibres underneath. Several tears involving peritoneum only were seen on the posterior aspect of the uterus, running downwards from the posterior edge of the tear for 2 in. or 3 in. Seen from within, the edges of the tear were infiltrated with blood, and some blood-clot still adhered to them.

REMARKS.

The specimen illustrates an exceedingly rare variety of rupture of the uterus—viz., a complete transverse rupture limited to the fundus, in contrast to the ordinary varieties, which begin in the lower uterine segment. No satisfactory reason for the occurrence of the rupture can be given; there was no cicatrix from a previous Cæsarean section, nor any gross abnormality (such as the presence of fibroids); also there was no pelvic contraction or abnormality in the presentation. Further, the foetus was a normal, full-term child. No degeneration of the uterine tissues was found on microscopical examination. Dr. R. D. Maxwell, obstetric registrar at the London Hospital, was inclined to think that the arrangement of the muscle fibres was more one in somewhat parallel lamellæ in the neighbourhood of the laceration than was the case in other parts of the same uterus.

Dr. DRUMMOND MAXWELL suggested that an unusual arrangement of muscle fibres might have predisposed to the rupture at the edge of the tear. Microscopic sections showed the muscle bundles running parallel to the plane of the laceration, and several planes of cleavage between these parallel bundles could be seen running out from the cavity. No lacerated muscle bundles could be seen, and the condition seemed to be rather due to cleavage of muscle bundles than their actual rupture. The possibility of a thinned placental site predisposing to the laceration was negatived by the fact that the wall was no thinner at the site of rupture than elsewhere in the cavity, and no villi were demonstrated in the uterine aspect of the laceration. There was no distinction between upper and lower uterine segments to be observed suggestive of a prolonged labour, nor any evidence of inflammatory change in the muscle (a myositis) which had been adduced as an explanation of these cases. Fatty degeneration of the muscle-cells was not demonstrated.

**Extensive Retroperitoneal Hæmatocele complicating a large
Uterine Fibroid: Operation: Cure.**

By WILLIAM ALEXANDER, M.D.

A SINGLE lady, aged about 50, connected with a teaching sisterhood, consulted me on May 8, 1909, about an abdominal tumour. The abdomen was very large, measuring at the most prominent part 40 in. in girth, and was occupied by a smooth solid tumour that had grown gradually for several years without any distressing symptoms and without interfering with a strenuous life. Lately, however—that is, for the past fortnight—the breathing was interfered with and the weight of the tumour was felt to be a burden. She became weak and exhausted and unable to carry out her duties. The menses were normal and had always been regular, and both the bladder and bowels were normal all through. Lately the abdomen had become somewhat tender, and her condition was approaching one to which the word “miserable” might soon be applied. The tumour occupied the middle line, was freely movable, and in the absence of menorrhagia, &c., was diagnosed provisionally as probably a solid ovarian. Owing to the objections of the patient, we were unable to make a vaginal examination. This was of the less importance as an operation was evidently required, and the exact nature of the tumour would be declared by the exploration, and could be dealt with just as well, no matter what the nature of the tumour might be. We easily satisfied ourselves that the tumour was not physiological, and recommended an operation as soon as convenient.

OPERATION.

Four days afterwards I opened the abdomen and found the tumour to be a large solid fibroid, which at the lower part dipped into the pelvis and originated in the fundus uteri. The upper part of the tumour very nearly reached to the ensiform cartilage. Its weight was afterwards found to be 12 lb. The tumour seemed to be very far forwards, projecting at an abnormal distance from the spinal column, but this when noticed was ascribed to sterilized pads that were used to keep the intestines well out of the field of operation. The broad ligaments were tied off, both ovaries being left behind, and the uterus was removed in

the usual way, the peritoneum being closely stitched over the end of the cervical stump.

Enormous Hæmatocele found.—On removing the sterilized pads we found a mass behind the intestines between them and the spinal column, and we could see through the translucent peritoneum that the mass consisted of a dark layer of blood fully an inch in thickness spread over the front of the spinal column and the region on each side. The hæmatocele—for such it was—went up between the layers of the mesocolon and for some distance between the layers of the mesentery, even up to the hilum of the liver. It diminished in thickness towards the pelvis, but existed over the sacral promontory. At the time the hæmatocele was first discovered, the broad ligaments did not contain any blood, but gradually and slowly filled up, the blood-supply coming down behind the peritoneum from the enormous hæmatocele that existed above. The peritoneum covering the posterior wall of the abdomen was so tense from the pressure of the blood beneath it that we were afraid the thin serous membrane might rupture and a gush of blood take place that would be immediately fatal. I placed a glass drainage-tube in the right broad ligament, bringing it out of the lower end of the cœliotomy wound, and closed the abdomen as expeditiously as possible. The anæsthetist now informed me that the pulse was good, and only accelerated by a few beats from what it was at the beginning of the operation. It was then 100, and was now 108. The comparatively rapid pulse we had noticed before operation was ascribed to nervousness, but we now felt inclined to attribute it to a pre-operative rupture of a vein, and to a gradually increasing venous hæmorrhage that had probably been going on slowly for two weeks before operation.

I saw the patient at the end of this year (December 28, 1909). She had been working strenuously for the past three months. The cicatrix was good; a minute sinus closed by a small granulation showed the spot where the tube had been. The abdomen was now quite flat, and no evidence of the hæmatocele existed. The contour of the front of the spinal column could be easily felt.

REMARKS.

Pelvic hæmatocèles of a certain size and position are comparatively common, but are mostly limited to the broad ligaments. They are usually connected with a ruptured extra-uterine tubal gestation. These hæmatocèles are sometimes post-operative, the result of the puncture of

a vein in the broad ligament by the needle that carries the ligature. This danger is now so well understood that it hardly ever occurs, and its occurrence would indicate an operative fault of great rarity and one not difficult to avoid.

When we opened the abdomen the tumour filled the whole space and was very prominent. The intestines were covered with sterilized dabs and seemed rather prominent, but nothing was thought of that till the uterus was removed. The peritoneum had been stitched together over the cervical stump, and we were proceeding to remove the gauze dabs when we found the intestines were pushed forwards and that there was a mass of something behind the peritoneum. Through the peritoneum we could see a lake of blood, homogeneous in appearance and very tense, whether quite fluid or not we could not say, but probably measuring several pints in quantity. The liver and stomach lay away behind in a deep valley. The crest of the swelling was on a level with the colon. That we might have some connexion from without with this sea of blood, I passed a glass drainage-tube into the right broad ligament, which was then empty of blood. A small quantity of blood came from the tube for the first few days. Gauze was then substituted. The aseptic condition was fortunately maintained throughout, as a septic condition of that mass of blood would have had rapidly fatal effects. One danger of sepsis lay in the operative technique, but the proximity of the intestines to the hæmatocele was another source of danger had the bowels been troublesome. The intestines, however, kept their germs at home, and the blood kept pure and was reabsorbed. The blood was not nearly absorbed by June 17, 1909, when she passed from under my care. In December, when I last saw her, the hæmorrhage had entirely disappeared. In my opinion the hæmorrhage had been very gradual and had occurred during the fortnight previous to operation. None had occurred after the operation. The regular, austere, clean life of the patient was a great factor in her recovery. As to the mechanism of the hæmorrhage, we can only surmise the rupture of a distended vein, similar to the ruptures that occur in varicose veins of the leg and the effusion of the blood behind the peritoneum.

We have not been able to find a similar case recorded. Probably there are such, as the other circumstances of the case are common enough, and we hope some references may be obtained from the audience this evening.

The position of the operator when he discovered the presence of the effused blood was not an enviable one, and the operation, which had

proceeded so far successfully, seemed all at once doomed to failure. The friends were told that recovery was almost impossible and that the end might be any time. It is probable that the insertion of the drainage-tube into the broad ligament was not necessary, nor perhaps advisable, and was more likely to do harm than good. It was used from a feeling that we were thereby in touch with the "enemy," and was used on the spur of the moment when it seemed most desirable to have the patient safe in bed before the inevitable collapse occurred.

The PRESIDENT said the largest pelvic hæmatoma he had ever seen had a traumatic origin. A young married woman was about to sit down on a chair, when a friend in play drew it back, and she fell rather heavily to the ground. All the symptoms of shock and severe hæmorrhage followed, and when he saw her, some forty-eight hours afterwards, the pelvis was full of blood and the area of dullness extended from one iliac crest to the other. The expectant plan of treatment by ice, externally and internally, was followed. She recovered, but there was a state of invalidism for some three years. This was years ago, but now the important question in many cases of hæmatoma, hæmatocele or other, was that of interference. It was often a nice point, as between the risk of suppuration and sepsis, where, as in the case he quoted, suppuration had not occurred. He referred to true hæmatoma, *not* hæmatocele. He understood from Dr. Alexander that there had not been any recent injury in his case. Cases of extensive hæmatocele from rupture of varicose veins in the broad ligaments had before been recorded in the Obstetrical Society.

Obstetrical and Gynæcological Section.

March 10, 1910.

Dr. H. MACNAUGHTON-JONES, President of the Section, in the Chair.

Squamous-celled Carcinoma of a Dermoid Cyst of the Ovary.

By ERNEST H. SHAW.

CLINICAL HISTORY: Mrs. H., aged 47, was admitted into the Great Northern Central Hospital under the care of Dr. Blacker on February 10, 1910. She complained of pain in the lower part of the abdomen. The patient has been married twenty-one years; has had no children or miscarriages. Menstruation began when aged 16, has always been regular, and lasts five days. Catamenia ceased three months ago. Two years ago the patient attended King's College Hospital for "appendicitis." She was told that she had a tumour, and was advised to have it removed. She refused to have this done. Patient remained quite well until Christmas, 1909, when intermittent attacks of pain in the right side of the abdomen started. During the past three weeks the pain has been constant, worse at night, and preventing the patient from sleeping. The pain is described as being like the "digging of a knife" in the side, and shoots down the right leg. A slight leucorrhœal discharge is present; the bowels are rather costive; micturition is sometimes difficult. The woman is thin and pale, and her appetite is poor.

Examination by Dr. Blacker (February 15): The abdomen is flaccid; a tumour, apparently solid, can be felt rising up half-way to the umbilicus, filling up the pelvic brim. There is probably some adherent intestine in front. *Per vaginam* the cervix is displaced forwards and to the left. The posterior fornix is partly obliterated by a softish tumour

attached to that felt in the abdomen; it feels cystic. The body of the uterus is displaced forwards, and its fundus can be felt in front of the cystic swelling. The tumour in Douglas's pouch appears attached to the uterus, but feels too soft for a fibroid.

Operation (February 20): Dr. Blacker opened the abdomen in the middle line; the omentum was found adherent to the anterior abdominal wall. The adhesions were broken down and the uterus exposed; it appeared to be fairly healthy. Behind the uterus was a large mass in the pelvis covered by coils of adherent intestine. The adhesions were separated with some difficulty and a cystic body exposed. In attempting to enucleate the cyst, its wall ruptured, and a large amount of purulent fluid and sebaceous material escaped. The cyst was separated with great difficulty owing to the dense adhesions to the intestines and broad ligament; a ragged mass of growth was attached to the deeper part of the cyst wall. The cyst originated in the left ovary; bleeding was slight. A gauze drain was inserted into the pelvis through the vagina and the abdominal wound closed.

The patient experienced a slight rise of temperature a few days after the operation, but this soon subsided after the discharge of a large amount of dark-coloured fetid material from the vagina. She is now well.

In its empty and collapsed condition the cyst measures about 4 in. in diameter. It is rounded in shape; the wall is thin above and thick lower down, and apparently fibrous in structure; adhesions are present on the surface. A circular mass of ragged, solid growth projects from the external surface of the lower part of the cyst; the growth is pale, yellowish white in colour, and is apparently malignant in character. A section through the growth and cyst wall shows the growth passing through to the inner aspect of the cyst, where it forms a flat, nodular mass about 2 in. in diameter. The cyst is partly divided into two portions by a thick band of tissue; the base of the latter is covered by a skin surface, from which are seen projecting a number of hairs which are almost white in colour; the inner surface of the cyst is rough, like corrugated skin, over a large area. Several masses of light-brown hair bound together by sebaceous material lay loose in the cavity of the cyst.

Microscopic examination of the growth shows typical squamous-celled carcinoma, with many "cell-nests" and much cornification of the epithelium. A section of the cyst wall and nodular mass seen internally shows the same type of growth, which is also continuous with the external tumour.

Conclusion: The cyst is a dermoid, and the squamous epithelium has undergone malignant changes. The growth has then passed through the wall of the cyst and formed the large malignant tumour there seen. Secondary inflammatory changes have caused the formation of the extensive adhesions found on the external surface of the cyst wall.

DISCUSSION.

Mr. TARGETT said he had operated on a similar case. A lady, aged 45, came under observation for a pelvic tumour causing difficulty in micturition. The uterus was pushed upwards and forwards by a rounded tumour, which filled Douglas's pouch and extended up half-way to the umbilicus. At the operation it was found to be a dermoid cyst of the left ovary, which was densely adherent to the back of the uterus, rectum, and pelvic wall. The operation was very difficult, and panhysterectomy was needed to control the bleeding and to get at the deep adhesions. The wall of the cyst was much thickened at one part, and the interior of the cyst exhibited a new growth which had the characteristic structure of a squamous-celled epithelioma with cell-nests. The patient made a good recovery, and remained in fair health for some months, until recurrence took place in the pelvic cavity. She died thirteen months after the operation, having suffered severely from invasion of the sacral plexus and from incontinence of urine and fæces. On another occasion, he (Mr. Targett) had performed an exploratory laparotomy on a single woman, aged 34, for an abdominal tumour which clinically seemed to be a malignant ovarian cyst. This was confirmed by the operation, and growth was found fungating through the capsule of the cyst, while secondary nodules had formed in the omentum and upon the anterior abdominal wall. No attempt at removal was made. Microscopical examination of a small nodule from the omentum showed that it had the structure of a squamous-celled epithelioma. From this fact it was presumed that the ovarian cyst was a malignant dermoid tumour. The patient died a few weeks after leaving the hospital, but no autopsy was obtained.

Dr. BLACKER at the time of the operation thought that the mass adherent to the uterus was inflammatory and not malignant, and the real condition was only recognized when the tumour was examined after removal. In view of the fact that there were a large number of adhesions, and that the peritoneum was involved, he had not thought it advisable to perform any further operation. At no time had the patient presented any symptom of carcinoma of the uterus, and the cervix, as far as it could be examined without dilatation, was quite healthy. At the present time the woman was quite free from any vaginal discharge.

A Case of Actinomycosis of the Ovary.

By ERNEST H. SHAW.

CLINICAL HISTORY: Miss B., aged 38, was first seen by Mr. Mower White in August, 1908. She was then suffering from a typical attack of appendicitis, and a large swelling was present in the right iliac fossa. The swelling subsided slowly and never completely disappeared. On September 28, 1908, the appendix was removed, together with a small abscess in connexion with it; the wound was drained and healed with a little suppuration along the track of the drainage-tube. In July, 1909, the patient was seen again. There was a large, tender swelling occupying the left half of the pelvic cavity; it formed a prominent tumour in the left hypogastric region and extended to the bottom of the pouch of Douglas, bulging into the rectum and vagina. On July 23 Mr. Mower White removed the tumour shown by an incision through the lower end of the left rectus abdominis. It lay in a suppurating cavity and was attached externally (lateral wall of pelvis) only.

The patient was very ill at the time of the operation, and died six days later from septicæmia. She had resided in North London, and had been employed for many years as a gold embroiderer. At the time of her death she was forewoman of the workroom. The place of employment was in the West End of London.

The tumour is roughly the same shape as an ovary; it measures about $2\frac{1}{2}$ in. by $2\frac{1}{2}$ in. by 2 in. in its chief diameters. The surface is covered by loose adhesions, through which round, yellow nodules, about $\frac{1}{4}$ in. diameter, are seen in the substance of the mass. On section the tumour is seen to be riddled with small abscesses, which contained a yellow "slimy" material. Several larger abscesses are seen near the surface. Bands of semi-gelatinous fibrous tissue traverse the whole section between the abscesses.

Microscopic examination shows many small abscesses, in some of which typical colonies of actinomyces are seen. No ovarian tissue is visible. The primary seat of disease was no doubt in the vermiform appendix. The ovary was infected by the actinomyces by extension from the appendix.

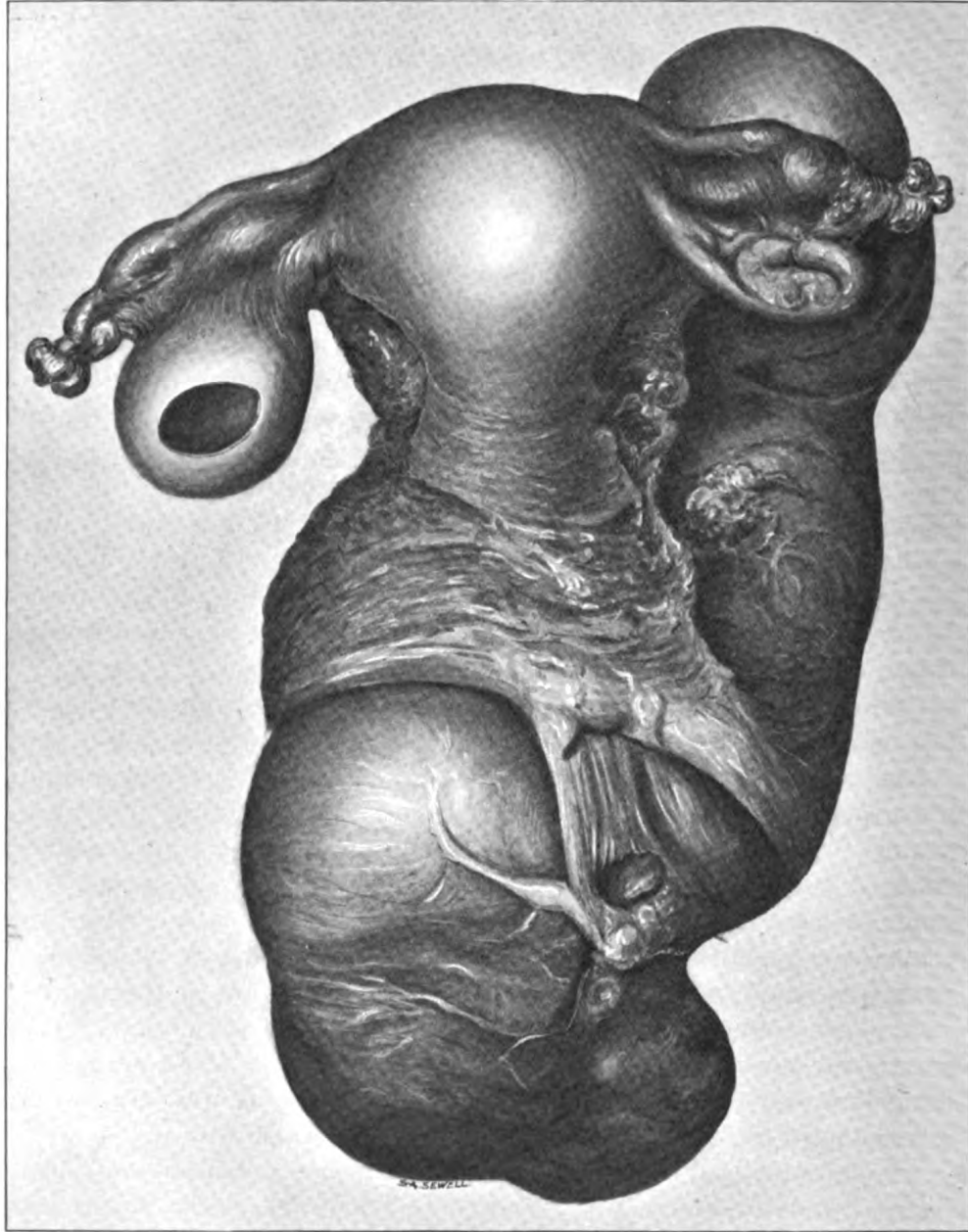
A Degenerating Fibromyoma of the Cervix.

By FREDERICK McCANN, M.D.

THE specimen was removed in August, 1909, from a lady aged 42. When she presented herself for examination on August 20, 1909, she complained of having a profuse foul vaginal discharge, irregular hæmorrhage, and incontinence of urine. Further, she had pain referred to both iliac regions and over the sacrum, which extended down the thighs. The discharge had become exceedingly fœtid during the previous month, although it had been slightly so since its onset. The irregular bleeding had existed for two months. She had had two children, the youngest being aged $6\frac{1}{2}$. For two or three months gradual wasting had supervened. She had worn a ring pessary for thirteen years, which was changed at intervals, but more frequently during the last year. Indeed she was supposed to have prolapse of the womb until it was discovered by another medical man that a more serious condition existed, and a consultation was advised.

When seen by me she looked very ill and anæmic, and had evidently lost flesh. On abdominal examination a hard mass could be felt in the pelvis, whose contour was difficult to define. On vaginal examination a copious thick, fœtid, brownish-yellow discharge issued from the vaginal orifice, and on introducing the fingers a globular œdematous softened swelling the size of a tennis-ball occupied and distended the upper segment of the vagina, apparently springing from the cervix. In the vaginal vault there was felt an annular ulcerated area, which bled readily on touch and which was causing marked thinning of the vaginal wall in this situation. The os uteri was difficult to identify. *Per speculum* a wash-leather slough was seen to cover the lower surface of the globular swelling. The mass felt *per hypogastrium* was now found to be situated in front of the uterus and to be movable with it.

Two days later the abdomen was opened by a median sub-umbilical incision, and the uterus was found to be situated posterior to a hard irregular mass, pushing up the peritoneum between uterus and bladder and extending into the bulging cervical growth. The appendages were separated and the vessels ligatured. The utero-vesical peritoneum was incised and the uterine vessels exposed, caught, and tied. Care was taken to prevent blood loss, owing to the anæmic state of the patient. The



Degenerating fibromyoma of the cervix. The drawing represents a posterior view of the uterus, appendages and upper portion of the vagina removed together with the tumour. The rounded cervical growth is seen hanging downwards, and the mass which lay between the bladder and uterus has a dome-like upper extremity which projects above the level of the right Fallopian tube. The left ovary contained a small cyst.

upper portion of the vagina was exposed after thorough separation of the bladder, and was incised, and the uterus and tumours removed. The operation was difficult owing to the extreme vascularity of the growth. Special care was taken to protect the pelvic cavity and the abdominal walls from the discharge issuing from the cervical growth. The pelvis was carefully covered in with peritoneum and the abdominal wall sutured in layers. The patient made an uneventful recovery, the wound healing by first intention. The specimen (see figure) consists of the uterus, appendages, and a portion of the vagina. The rounded tumour, previously described, is attached to the anterior lip of the cervix and is continued into an elongated nodular mass lying between the uterus and bladder. A section bisecting the uterus and tumour shows that the structure of the tumour, white and fibrous, resembles that of a fibroid. At the upper and lower poles there is evidence of increased vascularity, whilst necrosis exists in the latter situation. Sections obtained from the portion of the growth projecting into the vagina show that it is a degenerating fibromyoma.

As has been already stated in the clinical history, it was assumed that she was suffering from prolapse, and a pessary had been put around the neck of the vaginal tumour, and as a result of continuous pressure an annular ulceration was produced. The condition of the patient, her rapid loss of flesh, and the dark-blue vascular condition of the tumour when removed suggested the possibility that the disease was sarcomatous. This, however, was not supported by the microscopic examination. Further, the improvement in her general condition has been remarkable, and she is now, six months after the operation, in good health.

The Pathology Committee reported that the specimen was one of degenerating fibromyoma of the cervix uteri.

A Case of Tubo-abdominal Pregnancy.

By FREDERICK McCANN, M.D.

THE specimen was removed from a woman, aged 32, who had been married for six years, and had not been pregnant. She had always menstruated regularly, the last period being in the first week of July, 1907. In the middle of August, 1907, she was seized with severe pain in the hypogastrium, accompanied by sickness, vomiting, and faintness, and some bleeding from the vagina, which lasted two hours. She

remained in bed for five days. Four days later the severe pain recurred, accompanied by sickness as before, with discharge of bright blood and "pieces of flesh" from the vagina, which was followed later by an offensive brown discharge. She was removed to a cottage hospital, where she remained seven weeks. During this time the offensive discharge continued, with occasional loss of blood, but the pain diminished. After leaving the cottage hospital she suffered from pain in the left side of the lower portion of the abdomen, which extended down the left leg. Her abdomen was gradually enlarging, her breasts swelling, and she suspected she was pregnant, although, owing to the passage of a "piece of flesh," she was informed that she had aborted.

The pain in the left side continued to increase before her admission into the Samaritan Hospital. When admitted this pain troubled her chiefly at night, and she had noticed she was becoming more constipated. Abdominal examination revealed an elastic, movable, painless tumour, regular in outline, partly solid and partly fluid, occupying the left half of the abdomen below the umbilicus. It felt about the size of a foetal head. Bimanually the swelling was found to extend into Douglas's pouch and to occupy the left half of the pelvis, pushing the slightly enlarged uterus forwards and to the right. Over the lower pole the outline was irregular, and marked pulsation was noted. Any attempt to move the tumour caused pain. A diagnosis of extra-uterine pregnancy was made, and operation recommended.

On November 20, 1907, the abdomen was opened by a median sub-umbilical incision, exposing a dark-purple thin-walled sac on the left side, to which intestine and a large sheet of omentum adhered. The adhesions were separated, the omentum requiring several ligatures. As at one part of the sac, close to the left Fallopian tube, there was evidence that a layer of peritoneum was covering it, this was incised, and, on attempting to detach it, the extreme thinness of the sac wall was discovered and an opening accidentally made into it. Out of the opening a quantity of bright blood gushed. Forceps were at once placed on the ovarian vessels, close to the bowel, and on the left uterine cornu. The sac was then rapidly separated and removed. The placenta was attached to the posterior surface of the uterus, the bottom of the pelvis on the left side, and the sigmoid colon. The edges of the raw surface on the bowel where the placenta was attached, and which appeared to be formed of thickened fibrous tissue, were inverted and united by catgut sutures. The meso-sigmoid was observed to be plentifully supplied with vessels to nourish the placenta. On the posterior

aspect of the uterus a large vessel was seen running upwards to the placental attachment. This vessel was secured by passing a needle under it, armed with a ligature. Where the placenta was attached to the uterus the other bleeding points were tied, and the raw surface covered by drawing the left Fallopian tube and round ligament over it and fixing with catgut. At the bottom of Douglas's pouch, on the left side, there was considerable venous oozing, and, as it was not considered safe to control it by stitching, a gauze drain was introduced, and was subsequently removed in forty-eight hours. The abdominal wall was united in layers; three silkworm gut sutures, left long, were united after the gauze was removed, in order to complete the closure of the abdominal incision. The operation was well borne, and the woman made a smooth recovery. The specimen consists of the placenta and amniotic sac, containing a four-and-a-half months' fœtus in good preservation. The fœtus had probably escaped from the Fallopian tube during the first attack of severe pain in the middle of August, and the placenta had subsequently become implanted on the sigmoid colon, the posterior wall of the uterus, and the pelvic floor. The increased vascular development in the meso-sigmoid was a marked feature, as well as the development of a large vessel on the posterior uterine wall.

The clinical history serves to emphasize the importance of avoiding what is not an uncommon mistake—namely, inferring that the passage of a decidual cast, accompanied by hæmorrhage, is a uterine abortion.

A Secondary Implantation Teratomatous Cyst.

By VICTOR BONNEY, M.D.

THE specimen shown was removed from a patient aged 26. In August, 1909, a large cystic tumour of the right ovary, and of a reddish colour, had been removed by Dr. Comyns Berkeley. The left ovary was then healthy, and no other tumour was present. In January, 1910, the patient returned to the Middlesex Hospital with a large tumour in the abdomen. At the operation this was found to consist of a cystic mass imbedded amongst, and indissolubly adherent to, the intestines. It was of a purple-red colour, and, though adherent, appeared definitely encapsuled. The left ovary was healthy, as was the stump remaining from the previous operation; nor were there signs of any other secondary growth. The tumour was removed, together with about 1 ft. of small

intestine and some 4 in. of sigmoid colon, to which it was adherent. Two end-to-end anastomoses were performed, and the abdominal wound was closed. The patient made an uninterrupted recovery.

The interest of the specimen lies in the fact that the original tumour of the right ovary showed the typical structure of a cystic teratoma, exhibiting various types of epithelium, masses of cartilage, and tooth germs, with which the structure of secondary growth entirely agrees. Its isolated position—not in the mesentery but amongst the intestines—the fact that no other signs of secondary growth were present, and the circumstance that the left ovary was healthy, all point to an origin by implantation from the primary tumour of the right ovary. Probably in removing this a small portion must have become detached, and, falling into a recess between the coils of intestine, became ingrafted there.

Abscess of the Ovary probably due to Infection by the *Amœba coli*.

By H. WILLIAMSON, M.B., and J. D. BARRIS, F.R.C.S.

THE patient was a married lady, aged 25, who had lived in India and had suffered from dysentery for three years. She had been married for four years, and had one child three years ago. For eighteen months she had complained of severe pain over the right iliac fossa, and severe pain in the rectum during defæcation. Menstruation had been regular in time, but the loss was greater than formerly. On examination a tender swelling, the size of a small orange, was felt in the right posterior quarter of the pelvis, displacing the uterus forwards and to the left, and apparently adherent to the anterior aspect of the rectum; this was thought to be an inflamed and adherent ovarian cyst.

Dr. Williamson advised removal of the tumour, and performed the operation a week after first seeing her. The abdomen was opened by a median incision, many adhesions were encountered, and coils of intestine were found adherent to the uterus and tumour, roofing in the pelvic cavity. After separating these it was found that the tumour was densely adherent to the posterior aspect of the uterus and to the anterior aspect of the rectum. It was separated, partly by the finger and partly by the knife, and together with the adjacent tube was removed. The patient made a good recovery.

After removal, the tumour was proved to be the ovary containing an abscess filled with greenish pus, sterile on culture, but containing large, coarsely granular cells, with a centrally situated nucleus, resembling the *Amæba coli*. Subsequently similar cells exhibiting amœboid movements were found in the fæces.

The specimen shown to-night is the right ovary laid open to show an abscess cavity at its lower pole. The cavity measures $\frac{3}{4}$ in. in diameter, and is lined by a thick corrugated membrane. On the outer surface of the ovary are a number of peritoneal adhesions.

Microscopical examination: The abscess wall is lined by degenerate connective tissue. No cells which can be identified as lutein cells are seen.

Dr. CUTHBERT LOCKYER said that the specimen shown by Mr. Barris had all the appearances of a typical lutein abscess. Its corrugated lining and the pale-yellow convolutions in the wall limiting the cavity were sufficient to show that the suppuration had occurred within a corpus luteum. He suggested that the specimen be referred to the Pathology Committee.

Carcinoma of the Body of the Uterus, with Keratinization of the Growth.

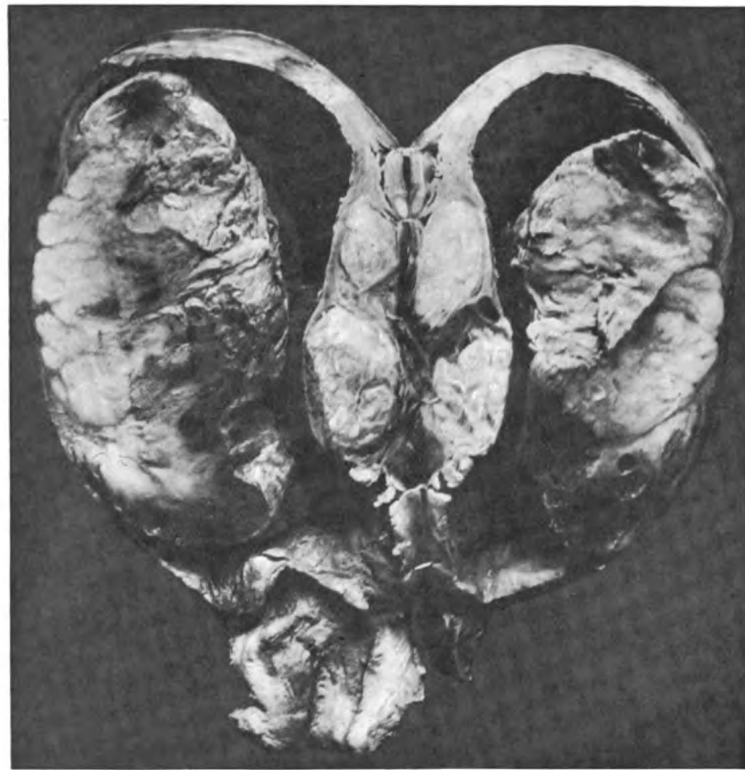
By J. H. TARGETT, M.S.

THIS specimen was removed from a single woman, aged 75, who had always enjoyed good health until about twelve months previously, when she began to be troubled with a foul vaginal discharge. Pain, at first absent, gradually developed, and during the last six months had been very severe and paroxysmal. The discharge was sometimes blood-stained, but there had been no serious hæmorrhage. Abdominal hysterectomy was performed, but the patient died on the thirteenth day from septic infection.

Description of specimen: The specimen consists of the whole uterus removed by operation. The body of the uterus is uniformly enlarged to the size of a three months' gestation. The cavity measures about 4 in. in length, $3\frac{1}{2}$ in. from side to side, and $2\frac{1}{2}$ in. from before backwards. The uterine cavity is more than half filled with a sloughing new growth which springs from the anterior wall of the uterus and projects into the cavity. The growth is sessile, but with a decidedly constricted

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attachment, so that while it measures $3\frac{1}{4}$ in. in its chief diameter, the base of the growth is barely $2\frac{1}{4}$ in. across. The cut surface of the tumour is white and friable at its base, where the wall of the uterus is invaded and the sinuous outline of the growing edge is remarkably distinct. It will be seen to have penetrated the wall so deeply that only a very thin layer of muscle separates it from the serous membrane. The superficial portion of the tumour is necrotic and stained dark from numerous



Carcinoma of body of uterus, with keratinization of the growth.

hæmorrhages into its substance. The lining membrane of the rest of the uterine cavity is for the most part smooth, but at one spot on the posterior wall it is raised over a small calcified fibroid, and near the cervix uteri the endometrium is superficially ulcerated where it has been in contact with the necrotic portion of the tumour. There are no nodules of secondary growth upon the external aspect of the specimen.

On microscopical examination the base and growing edge of the tumour have the structure of a columnar-celled carcinoma with a distinct tubular arrangement of the columnar cells. But sections taken from the body of the growth at some distance from the base are remarkable for the abundance of "cell-nests" and masses of keratinizing epithelium. These bodies have a considerable resemblance to the cell-nests of a squamous-celled epithelioma, and the elements of which they are composed are somewhat concentrically arranged. The growth around them consists of small spheroidal carcinomatous cells which are evidently due to rapid proliferation, and all traces of the original tubular structure are lost. Thus the specimen is of interest not only on account of the unusual shape and relations of the tumour, but also because of the presence of so much keratoid degeneration in an otherwise rapidly growing columnar-celled carcinoma. The accompanying photograph represents the uterus as laid open by a median sagittal section. It shows the constricted attachment of the main tumour to the anterior wall of the uterus, and some small fibroids embedded in the posterior wall. The cavity of the uterus is considerably dilated and contained foul discharge. The cervix uteri was accidentally detached from the body, but has been sutured in place.

Adenofibromatous Polypus of Uterus showing Early Malignant Disease.

By J. H. TARGETT, M.S.

MRS. B., aged 33, never pregnant, was sent home from South Africa on account of severe menorrhagia. This had gradually developed during the previous twelve months and was not relieved by curetting. On arrival in England, the patient was very anæmic, and was suffering from persistent uterine hæmorrhage. On examination the uterus was somewhat enlarged, its outline smooth, and the cervix not dilated. With the evidence of severe hæmorrhage and some enlargement of the uterus, a diagnosis of intra-uterine polypus was made, and an exploratory operation was advised. The cervix dilated easily and a polypus the size of a walnut was felt. When seized with a vulsellum, the polypus rotated freely, showing that the pedicle was small. By cutting it up with scissors the tumour was easily removed from the uterus, and no other tumour could be felt in the uterine wall. On naked-eye examination the

fragments had a greyish appearance, and were somewhat friable, unlike the fibrous structure of the ordinary fibromyomatous polypus of the uterus. As a microscopical examination revealed what appeared to be a malignant growth, it was thought advisable to remove the uterus by abdominal panhysterectomy without delay. This was done a few days after the previous operation, and the patient made a good recovery. The uterine appendages were not removed. No evidence of growth was found in the abdomen, and sections of the fundus uteri at the site of the polypus showed no growth in the uterine wall.

Description of the microscopical section: The polypus has the general structure of an adenofibroma, such as would originate from the endometrium. The stroma consists of fibrous tissue, the elements of which are richly nucleated and arranged in interlacing bundles. One or two strands of unstriped muscle may be seen, but the stroma is chiefly fibrous tissue. The free surface of the polypus is covered with a layer of columnar cells, like those of the endometrium. Embedded in the fibrous stroma are numerous glandular tubules, having a distinct lumen and lined with columnar epithelium. In close connexion with some of these tubules are clusters of epithelial cells, which at once arrest the attention and raise the suspicion of malignant disease in an early stage. These clusters interrupt the uniform columnar-celled lining of the tubules, and then project as buds, either into the lumen of the tubule or into the adjacent fibrous stroma. In a few instances the site of a tubule is marked by an enlarged cluster, all the columnar cells having disappeared. Similarly one or two irregular masses of cells may be seen in the stroma, quite removed from any apparent connexion with a tubule, but obviously of the same type as the clusters above mentioned. In the smallest clusters the elements seem to originate from the basal cells of the epithelial lining, and, as they proliferate, the columnar cells are displaced inwards and gradually destroyed. These appearances are not due to any obliquity of the section, nor to an accidental folding of the tubular wall. Neither can they be explained as adenomatous buds of the glandular acini, because the clusters of cells are solid and show no tendency to the formation of a tubule. Consequently, I am of opinion that the condition represents the early stages of the development of malignant disease (carcinoma) in an otherwise simple adenofibromatous polypus.

The Pathology Committee reported that the specimen showed evidence of glandular epithelial proliferation, probably of a malignant type.

The PRESIDENT (Dr. Macnaughton-Jones) said that the only case of malignant degeneration in the centre of a uterine polypus he had ever seen was in the late Professor Rosthorn's clinic in Heidelberg. A polypus, apparently benign, had been removed, but on section Professor Schottländer had found malignant degeneration, sarcomatous in nature, within the growth.

Microscopic Appearances in Tubal Pregnancy.

By THOMAS CARWARDINE, F.R.C.S.

Mr. CARWARDINE gave a demonstration by the epidiascope of a series of microphotographs from a case of ruptured tubal pregnancy, specially prepared to exhibit certain features of the syncytium, notably the cell-processes, which are probably of considerable importance in the physiology and pathology of the embryonic vesicle. The specimen was taken from a patient, aged 29, who was operated upon about twelve hours after the rupture.

The earlier slides included sections through the Fallopian tube at the point of rupture and elsewhere, and the chorionic vesicle and sections from various parts of its wall were demonstrated, the membrane being thicker where the villi were most developed. Under higher magnification the general arrangement of the villi was shown, and the manner in which the investing epithelium is generally beset with cell-processes, to which attention was directed.

One specimen of a chorionic villus revealed the origin of multinucleated wandering cells from the foetal epithelium, having cell-processes also, which, where not coagulated together by the hardening reagent, were of a delicate silken character, embracing a red corpuscle at one place. Some of the wandering cells were attached, others free.

A section near the point of rupture showed indications of the "cell-sheet" [1], also numerous cells derived from the trophoblast invading the blood-clot and undergoing amitotic cell-division; and enlargements of these were thrown upon the screen. They resembled the invading cells seen in the tissues outside a chorion-epithelioma.

Villi in contiguity showed great diversity in the characters of the epithelial coverings: one might have a cap of squamous elements; another, columnar cells with brush-like, free edges; a third, varieties in epithelial type, from the cubical or columnar cell to irregular multinucleated protoplasmic masses—all having cell-processes. A typical

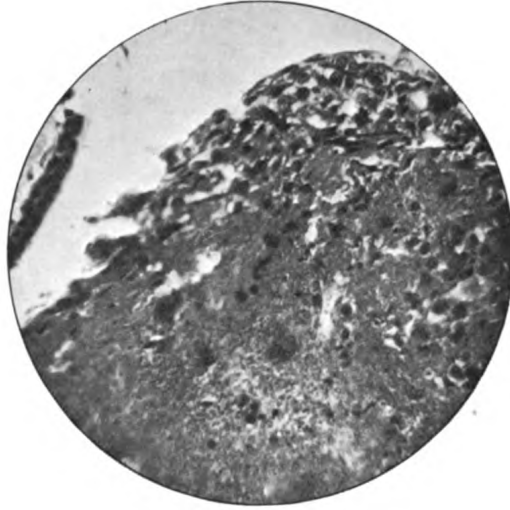


FIG. 1.

Section near point of rupture, showing the edge of a villus and indications of the "cell-sheet" to the left, and also blood-clot to the right, which is invaded by cells derived from the trophoblast, which are multiplying by direct division of the nucleus. Obj. $\frac{1}{8}$.



FIG. 2.

A chorionic villus in section, showing the foetal origin of wandering cells having cell-processes. Three such cells are attached above, and a portion of a detached cell is seen below and to the left. Obj. $\frac{1}{8}$.

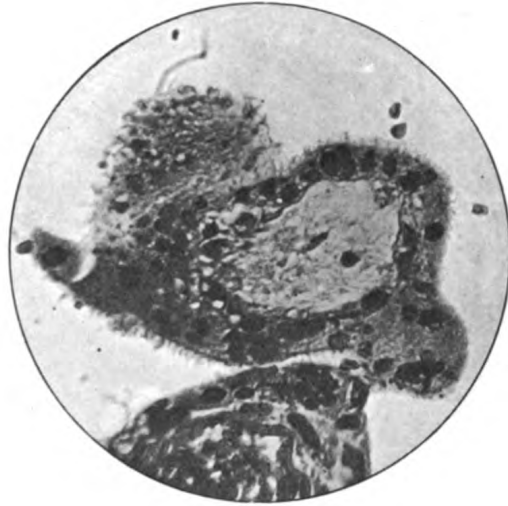


FIG. 3.

An enlarged microphotograph : shows irregular syncytial masses of protoplasm, having cell-processes, from which are derived the cells of Langhan's layer on the one hand, and masses of free nuclei and processes on the other hand. Obj. $\frac{1}{8} \times 3$. (Compare fig. 12, plate IV, in Teacher's monograph on "Chorion-epithelioma," *Brit. Journ. Obst. and Gyn.*, 1903, iv.)

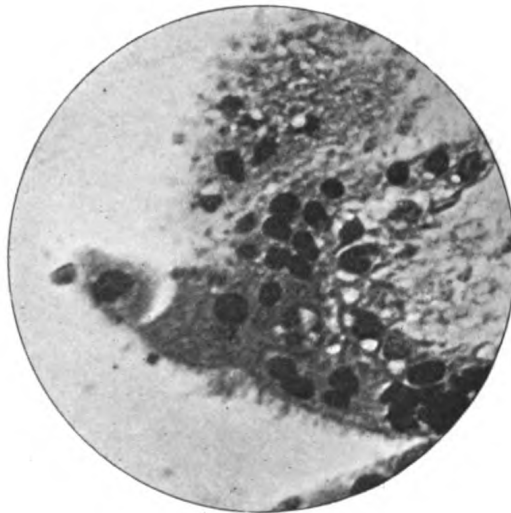


FIG. 4.

Portion of the previous figure more enlarged, to illustrate the origin of the mass of free nuclei with processes from the syncytium. The largest free nucleus is undergoing division. Obj. $\frac{1}{8} \times 6$.



FIG. 5.

Portion of fig. 2 enlarged, showing the origin of a wandering cell, with cell-processes, from the trophoblast. Obj. $\frac{1}{8} \times 3$.



FIG. 6.

Portion of the same specimen as fig. 2, enlarged, and showing a free wandering cell, having processes which are very delicate in character where they may have been less affected by the hardening reagent. Processes are also seen, cilia-like, on the surface of the chorion at the lower part of the figure. Obj. $\frac{1}{8} \times 3$.

illustration was given of such a section, which indicated the origin of Langhan's layer from the syncytial cells, and attention was drawn more particularly to the origin, from the syncytium, of masses of nuclei having numerous processes, the nuclei undergoing division. Such a mass is found free in the intervillous space, and in all probability has important significance, both in the physiological condition and in chorion-epithelioma; and in support of this suggestion a drawing in Teacher's monograph was referred to [2], a portion of which gave similar indications, though not clearly, to those in the photograph exhibited.

Some of the micro-photographs shown were under very considerable magnification, and much trouble had been expended in reproducing the sections, which had been cut and stained by Professor Walker Hall. A few of them are represented in the accompanying figures.

REFERENCES.

- [1] BERKELEY and BONNEY. "Tubal Gestation: a Pathological Study (illustrated)," *Archiv. Middlesex Hosp.*, 1905, iv, p. 1.
- [2] TEACHER. "Chorion-epithelioma," *Brit. Journ. Obst. and Gyn.*, 1903, iv, p. 98.

The PRESIDENT said that the Section was greatly indebted to Mr. Carwardine for the very beautiful series of slides which he had shown. They were of importance as showing certain pathological correlations between these tubal decidual appearances and those seen in chorion-epithelioma.

**Carcinoma of the Cervix of a Prolapsed Uterus in a Patient
aged 86, with a Contact Carcinoma on one of the Labia.
Vaginal Hysterectomy.**

By HENRY RUSSELL ANDREWS, M.D.

E. H., SINGLE, aged 86, was brought to see me in November, 1909. The menopause had occurred forty years previously, and since then there had been no bleeding until nine or ten weeks before I saw the patient. For five or six weeks there had been slight bleeding, and for the last four weeks the bleeding had been rather severe, especially at night. For four weeks she had noticed a very tender mass protruding from the vagina, and had experienced a great deal of pain and tenderness in sitting. There had been no disturbance of micturition or defæcation, and little, if any, wasting. The patient was a tough, hale, old countrywoman. The

cervix projected about 2 in. outside the vulva, and the fundus uteri was fairly high up. There was a foul carcinomatous ulcer, with deep excavation and a good deal of new growth, about the size of a half-crown, on the right side of the vaginal portion of the cervix, extending on to the vaginal wall. On the inner side of the posterior part of the right labium, where it was in contact with the diseased part of the cervix, was a carcinomatous ulcer about the size of a shilling.

I sent the patient in to the London Hospital, and operated a few days later. The operation was begun with the idea of removing the whole of the cervix and the involved part of the vaginal wall, but the tissues were so vascular that it was thought wiser to remove the whole uterus. Vaginal hysterectomy was performed as rapidly as possible. The body of the uterus was brought out retroverted and the broad ligaments tied from above downwards, the last step of the operation being the separation of the bladder. The first ligature applied to the right uterine artery cut right through it, and many bleeding points on the raw surface of the base of the bladder required ligatures. The wound was closed by suture of the peritoneum and the vaginal flaps, a small drainage-tube being left in, as there seemed to be a possibility of some oozing. The carcinomatous ulcer of the right labium was then excised. The operation lasted thirty-five minutes. The patient stood it extremely well; there was very little shock, and she called for a newspaper two days later so that she might follow the course of the debate on the Budget. There was no oozing, and the tube was removed on the third day. The patient made an uninterrupted recovery, got up on the eighteenth day, and left the hospital on the twenty-third day.

The uterus was 5 in. in length, the supravaginal portion of the cervix being much elongated. The body of the uterus contained several small fibroids. Microscopically both ulcers are seen to be squamous-celled carcinomata, though the appearances are not exactly alike, the contact carcinoma being made up of cells smaller than those of the cervix carcinoma, and possessing fewer cell-nests.

The case exhibits three points of interest:—

(1) Carcinoma of a prolapsed uterus. Although chronic ulceration is very common in cases of prolapse of the second or third degree, carcinoma is surprisingly rare. The patient had been aware of the prolapse for only four weeks, but it is impossible to believe that the very considerable elongation of the uterus which existed had been produced in so short a time. It is probable that the uterus had been prolapsed for a much longer period, as the presence of the contact carcinoma on the labium would seem to show.

(2) The patient's advanced age, 86. In spite of this the disturbance caused by the hysterectomy was very slight.

(3) The presence of a contact carcinoma. It is quite certain that the carcinoma had not spread by extension along the vaginal wall, as there was a clear area of healthy mucous membrane, 3 in. across, between the two ulcers.

Mr. Butlin¹ in his address in surgery at the annual meeting of the British Medical Association in 1907, referred to five cases in which carcinoma had occurred on one side of the vulva from contact with carcinoma on the other side, and to one case in which a columnar-celled carcinoma of the cervix had infected the vaginal wall, a patch of columnar-celled carcinoma being found on the vagina 3 cm. from the cervix.

DISCUSSION.

The PRESIDENT said that he had operated on a patient who had suffered from complete prolapse of twenty-five years' duration; the portio and adjacent vaginal surface in that case were eroded, but there was no malignancy. She was aged 74 at the time of the operation, some eleven years since, and never suffered from any subsequent local trouble. The rectum and bladder were included in the prolapsed sac.

Dr. MCCANN said that he had not seen an example of cancer of the cervix associated with prolapse of the uterus. He had, however, seen examples of "contact infection" in the labia and posterior vaginal wall. In the latter situation a polypoid cancerous cervical growth had produced infection at its point of contact with the vaginal wall, a portion of sound tissue existing between this contact growth and the cervix. It was difficult to be sure that contact infection was the correct explanation in many cases, for it was more probable that direct spreading along the lymphatics was the real cause. Indeed, what was most remarkable was that contact infection was so rare, even when all the conditions favourable for its occurrence were present.

Dr. BLACKER said he had seen one case of prolapse of the uterus in which there was a carcinoma of the cervix so extensive that the cellular tissue was involved to a considerable degree and the uterus could not be replaced. In view of the important part that chronic irritation had been shown to play in the causation of carcinoma, it was a most curious fact that carcinoma of the cervix was so uncommon in cases of prolapse. He had never seen any adequate explanation of this fact. Did Dr. Russell Andrews know of any explanation? Might it be associated with the very poor and sluggish circulation in the vessels of the cervix, which was a condition present in bad cases of procidentia uteri?

¹ *Brit. Med. Journ.*, 1907, ii, p. 255.

164 Andrews: *Case of Myomectomy during Pregnancy*

Dr. ANDREWS, in reply, agreed with Dr. McCann that it was possible that cases of "contact cancer" might be due to infection of lymphatics. He would have preferred to remove the whole of the vagina, but felt that this was unjustifiable, as it would have prolonged the time during which the patient must be kept under anæsthesia. The same remark applied to removal of glands in the groin, which he had carried out as a routine practice in all other cases of carcinoma of the vulva. Like Dr. Blacker, he found it impossible to explain the rare occurrence of carcinoma in prolapsed uteri.

A Case of Myomectomy during Pregnancy.

By HENRY RUSSELL ANDREWS, M.D.

A PATIENT, aged 23, was admitted into the London Hospital on August 31, 1908, with pregnancy complicated by a fibroid tumour of the uterus. After the birth of her first child, fifteen months before, her doctor found a fibroid, the size of a hen's egg, on the anterior surface of the uterus, and warned her that she might need to seek advice if she became pregnant again. Her last menstrual period had occurred nine weeks before her admission to the hospital. She was a healthy woman, and complained only of the ordinary symptoms of pregnancy and of a certain amount of cramp in the back and abdomen.

On abdominal examination a hard, rather nodular, movable mass was found, rising out of the pelvis to a point $1\frac{1}{2}$ in. above the umbilicus. On vaginal examination this hard mass was found to lie chiefly above the brim of the pelvis. The cervix was high up in front, while Douglas's pouch was filled by a soft, elastic swelling, which was evidently the body of the retroverted pregnant uterus. It was impossible to separate the hard mass from the body of the uterus. The diagnosis made was that of pregnancy of about nine weeks, complicated by the presence of a large subperitoneal fibroid, probably sessile rather than pedunculated, attached to the anterior wall and causing retroversion of the uterus. An attempt to push up the body of the uterus with the patient in the genupectoral position failed. From the fact that the body of the uterus was pressed down and fixed, with the fundus in the hollow of the sacrum, it was extremely probable, if not certain, that urgent symptoms would arise before long—i.e., retention of urine and abortion, on account of the inability of the uterus to rise up out of the pelvis. If an operation were performed at once it was almost certain that the uterus could be left

behind, so that, even if the removal of the fibroid caused abortion, the patient would be left with a uterus capable of performing its functions later on. If left until urgent symptoms appeared the risk of abortion would be greater, and possibly, at a later stage of pregnancy, myomectomy, apart from hysterectomy, might be impracticable.

It was decided, therefore, to open the abdomen and remove the tumour. A quarter of a grain of morphia was injected subcutaneously, half an hour before the operation, to diminish the chance of abortion. The operation was performed on September 4; an abdominal incision 8 in. long was necessary. A myoma-screw was inserted into the tumour, which was dragged out of the abdomen. The uterus left the hollow of the sacrum with a sucking sound, showing that it was pretty completely impacted. The fibroid sprang from the anterior wall of the uterus, reaching as low down as the utero-vesical reflexion of peritoneum. The pregnant uterus could be recognized behind, but the fundus was blended with the tumour, so that no line of separation could be seen. An incision was made round the tumour, about 3 in. away from the surface of the uterus, and the tumour was enucleated in a few seconds, leaving a bleeding bed, roughly circular, with a diameter of about $5\frac{1}{2}$ in. The hæmorrhage was very free; I put it down at about 1 pint, while Dr. Maxwell thought that 2 pints of blood were lost; possibly I was biased. Pressure-forceps were applied to the chief bleeding-points to control the hæmorrhage temporarily, and buried catgut sutures were inserted until the wound was almost flat and superficial. During the insertion of these sutures the raw area was diminished in size considerably by retraction of the uterine muscle. The wound was finally covered by a continuous catgut suture joining the vesico-uterine peritoneum to the peritoneal surface of the uterus above the wound, a transverse scar about 3 in. long being left. At the end of the operation the uterus had regained its normal position. The patient bore the operation very well, her pulse at the end being 108 to the minute; she was given a suppository of $\frac{1}{4}$ gr. of morphia and 2 pints of saline solution. Recovery was uninterrupted.

The patient left the hospital on September 25, twenty-two days after the operation. In February, 1909, she was delivered of a full-sized living child, the labour and puerperium being perfectly normal. The tumour removed weighed $4\frac{1}{2}$ lb.

I shall be interested to hear the views of Fellows as to whether they consider that this patient should have been left alone, except for catheterization if retention occurred.

DISCUSSION.

Dr. HERMAN thought there could be no doubt that Dr. Andrews had treated this case in the best possible way. He had given his reasons for deciding on the course to be adopted most clearly and convincingly.

Dr. HERBERT SPENCER said there was no doubt that the proper treatment had been adopted in this case, which had features excepting it from the general rule against performing myomectomy for corporeal fibroids during early pregnancy. It was not to be expected that so large a tumour would permit the uterus to rise out of the pelvis. He had seen a case where a tumour, nearly as large as a fist, which grew from the anterior wall, caused retroversion of the gravid uterus and by its movement, when palpated through the distended bladder, closely simulated ballottement of the foetal head. In that case the tumour, being much smaller than the specimen shown by Dr. Andrews, did not prevent the uterus from rising out of the pelvis, nor interfere with the pregnancy or labour.

Dr. MCCANN said that he had recently successfully enucleated a fibroid the size of a cocoanut from the uterus of a woman five-and-a-half months advanced in pregnancy. The operation was done on account of very severe *pain in the tumour*, which on removal presented the typical appearances of red degeneration. Myomectomy was the ideal operation when it could be performed, and he desired to draw special attention to three points in the technique. First, the importance of making the incision as near to the middle—avascular—line of the uterus as possible. Secondly, cutting within, and not into, the capsule of the tumour. Thirdly, plugging the cavity at once with gauze in order to arrest hæmorrhage until it was finally closed with sutures. By attention to these points much bleeding would be prevented during the operation.

Dr. ANDREWS, in reply, agreed with Dr. Spencer that in most cases of pregnancy associated with fibroids no interference was called for, and looked on pregnancy as being in itself a contra-indication to operation on fibroids unless they were causing pressure symptoms, impaction of the uterus, or severe pain. As Dr. Maxwell had noted, retraction was very marked in this case, so that the flaps, which looked redundant at first, were just sufficient to cover the raw surface without tension. He had used pressure forceps on spouting vessels rather than pressure with gauze, because he thought that the latter would have had to be applied with such severity that abortion would have been likely to ensue.

Obstetrical and Gynæcological Section.

April 14, 1910.

Dr. H. MACNAUGHTON-JONES, President of the Section, in the Chair.

The Place of Cæsarean Section in the Treatment of Placenta Prævia.

By HENRY JELLETT, M.D.

WHEN you, Sir, did me the honour of inviting me to read a paper before this Section of the Royal Society of Medicine on the place of Cæsarean section in the treatment of placenta prævia, you pointed out that of late considerable prominence had been given to this subject owing to the elaborate discussion of it in America by the American Gynæcological Society. You also suggested the propriety of this Section expressing some opinion as to the value of the operation in such a serious complication of pregnancy. I need not say how pleased I am to avail myself of this opportunity of placing my views before you, and, though my paper may not in itself prove a valuable contribution to the literature of the subject, I hope that, by introducing a discussion which will be of value, it may serve its purpose.

I should perhaps say at the beginning that, personally, I have had no experience of any form of Cæsarean section as a treatment of placenta prævia, and though at first sight this might seem a fatal objection to my writing on the subject, yet, on the other hand, I do not know of any one in this country who has had much, if any, experience of the method. Various gynæcologists and obstetricians may have performed Cæsarean section on patients with placenta prævia because of some other accompanying complication of the pregnancy, but I do not know of any one who, so far, has adopted it on account of the placenta prævia alone. We

are therefore, I think, discussing the operation with a view to determine, not the results of our individual experience, but whether we ought to have any such experience: whether, in other words, the operation has any place in the modern treatment of placenta prævia.

There are certain limitations which I wish to impose on this paper, though whether the subsequent speakers observe them or not is a matter entirely for themselves. In the first place I shall confine myself to a discussion of the value of Cæsarean section as a treatment of placenta prævia, and not as a treatment of certain cases of placenta prævia complicated by other conditions which in themselves call for Cæsarean section. The reason for this limitation requires no explanation. In the second place I propose to discuss this form of treatment from the standpoint of the specialist, and not from that of the general practitioner, because, whatever arguments I may bring forward in support of the operation from the former standpoint, I can only regard it as an inadmissible form of treatment from the latter standpoint. The undoubtedly high mortality of placenta prævia in the hands of the general practitioner can be reduced by very much more simple means than by the adoption of an operation which, in his hands, and under the conditions under which he must operate, brings in a new and very potent cause of mortality.

In consequence of these limitations my paper resolves itself into an answer to the question: Will Cæsarean section lessen the mortality of placenta prævia for the mother or for the child?

The first thing to be considered is the existing mortality, in order that we may know to what extent it is capable of improvement. I shall discuss the maternal mortality first. It appears to me, speaking from an experience based on the statistics of the Rotunda Hospital, that there is a rather exaggerated idea of the mortality caused by this condition. Whether this is the result of Lawson Tait's statement that the mortality of placenta prævia under obstetric methods of treatment amounted to 40 or 50 per cent., or of Mr. Bland-Sutton's implication that the condition is only slightly less serious than a mesometric pregnancy, or whether it is the result of experience of forms of obstetrical treatment other than those adopted in Ireland, I do not know. Whatever may be the reason, the Irish statistics do not seem to warrant us in regarding placenta prævia as a complication of pregnancy to which an excessively high rate of maternal mortality is necessarily attached.

I will take the statistics of the Rotunda Hospital for the last twenty years. During that time 32,546 women were confined in the hospital,

and of these 138 had placenta prævia. Five of these 138 women died, a percentage of mortality of 3·6. I shall, I believe, be able to show that, with a single exception, each of the women who died were either moribund from hæmorrhage or in a condition of advanced sepsis when admitted to the hospital; and, further, that in every case in which the regulation treatment was adopted from the first, the patient recovered.

A very similar rate of mortality at the hands of American specialists was recorded by Fry in a paper read at the April meeting of the American Gynæcological Society. He records 161 cases with five deaths—a percentage mortality of 3·1. As I understand his paper, the treatment adopted in these cases was identical with that adopted at the Rotunda Hospital—namely, bipolar version, bringing down of a foot, and leaving the expulsion of the child to the natural efforts. From these statistics, therefore, it appears that, so far as the mother is concerned, in only about 3·5 per cent. of cases is it necessary to look to Cæsarean section to improve our results.

I now present a table (p. 170) showing the nature of the fatal cases of placenta prævia that occurred at the Rotunda Hospital in the last twenty years, and the treatment adopted in them. From a study of it we shall be able to form some opinion as to the possibility of improving the results by the adoption of Cæsarean section.

Of the five cases enumerated in this table, we see that three (Nos. 2, 3, and 4) died of septic infection received before admission. I do not think that its most strenuous supporter could suggest that under such circumstances Cæsarean section would have been of any benefit, and, therefore, I propose to omit these cases from the number of those patients whom the performance of Cæsarean section might have saved.

There thus remain two cases—the first and the last. No. 1 occurred a considerable time ago—nearly twenty years. I cannot say why internal version was attempted instead of bipolar version, but presumably it was because the membranes were ruptured and the liquor amnii had escaped. It is also difficult to say whether the uterine rupture occurred during the attempt to turn the foetus or during its extraction with the forceps. I do not think that there is subsequently any record of a case that was similarly treated, and I do not think that the authorities of the Rotunda Hospital would, under similar circumstances, adopt this treatment again. Further, it does not appear to me to be a case in which any one would have advised Cæsarean section, although, if the rupture had been recognized before the application of the forceps, one might have done so. Under such circumstances the Cæsarean section

would have been performed on account of the uterine rupture, and not on account of the placenta prævia.

The last case, No. 5, is the only one on the list in which death occurred from hæmorrhage from the detached placenta; but in this case the patient was almost moribund on her admission to hospital. She

No.	Year	Name	Situation	Remarks
1	1890-91	M. F.	Marginal	Patient admitted with hæmorrhage; marginal placenta prævia was diagnosed, and an attempt made to perform internal version; this was abandoned in favour of the forceps, as the lower uterine segment was very thin; extraction was easy, but subsequently, during the removal of the placenta, which was adherent, rupture through the upper portion of Douglas's pouch was found, and the patient died on the couch
2	1898-99	R. C.	Marginal	Patient had driven nineteen miles to hospital; previously attended by a midwife, who had made frequent examinations; she had had intermittent hæmorrhage for two months; on admission, her vagina was enormously distended with clots and her clothing saturated; external version was performed and then a foot brought down; fœtus expelled in an hour; twelve hours later, pulse 160, temperature 103.4° F.; temperature later rose to 104° F., and patient died on third night after admission
3	1898-99	A. T.	Central	Patient admitted with history of repeated hæmorrhages and rigors; she had been examined by a "handy woman" and two doctors; on admission, pulse 140, temperature 96° F.; placenta and child expelled just after admission; six hours later, temperature 102° F., pulse 144; temperature rose next evening to 105° F., and continued so until seventh day, when it reached 106° F., and patient died
4	1903-04	A. S.	Central	Patient admitted with the vagina plugged, and in a very weak state; bipolar version was performed, and child expelled two hours later; on fourth day the temperature was 101° F., and on following day 102° F.; streptococci found in the vagina; died of acute sepsis on eleventh day
5	1905-06	E. C.	Marginal	Patient in a collapsed state; she had been bleeding for several hours, and during a long railway journey prior to admission had fainted four times; bipolar version was performed; the hæmorrhage completely ceased, but she died three hours after admission

was treated in the regulation manner by bipolar version, with the result that all hæmorrhage at once stopped; but, in spite of this, she died, undelivered, three hours later. Could Cæsarean section have made any difference? It could not have checked the hæmorrhage any more quickly, and it certainly would not have caused less shock than the bipolar version. Therefore I think the answer must be in the negative.

Thus, of the five patients who died, amongst the 138 patients treated for placenta prævia at the Rotunda Hospital during the last twenty years, there does not appear to be one who would have been benefited by the performance of Cæsarean section.

Why is this so? I shall try to state what appears to me the probable reasons in a few words.

The causes of death in placenta prævia in the vast majority of cases can be grouped under one or more of three heads:—hæmorrhage; sepsis; and shock, by which I mean the associated effect on the patient of hæmorrhage and of the nerve irritation caused by operative interference. What is the relative value of the treatment of Braxton Hicks and of Cæsarean section in each of these conditions?

If anything is clearly established about placenta prævia it is that the treatment of Braxton Hicks, when carried out in a skilful manner, immediately checks the bleeding from a detached placenta prævia, and that, if it can be carried out, no other treatment is necessary. Moreover, it appears equally certain that it is no more likely to be followed by sepsis or by shock than is Cæsarean section; indeed it would appear to be considerably less likely to be followed by such results. If the patient is already septic when she comes under treatment, dissemination of the infection is probably less likely after the treatment of Braxton Hicks than after Cæsarean section; although Cæsarean section might improve the prognosis if it was followed by a total hysterectomy, for a similar reason that hysterectomy is sometimes said to be the best treatment of profound septic infection of the uterus. This operation is a very formidable one, and, when performed on a patient weakened by previous hæmorrhages, would almost certainly be fatal, although, if the patient recovered from the immediate shock, the prognosis might be then improved. Such a case, however, hardly comes within the limits of this paper. If the patient is profoundly collapsed when she comes under treatment, Braxton Hicks' treatment, carried out under an anæsthetic, is surely less likely to aggravate shock than is Cæsarean section, provided, as I hope everyone now recognizes, it is not followed by immediate extraction of the foetus, and that delivery is left to the contractions of the uterus.

Where, then, is the place of Cæsarean section in the modern treatment of placenta prævia? If you accept my arguments, and if you regard the 138 cases treated at the Rotunda Hospital as representative of the general run of such cases, it appears that there is only one possible class of case in which Cæsarean section may be advisable—

namely, that in which it is impossible to carry out the treatment of Braxton Hicks.

There appear to be only three conditions which can make the treatment of Braxton Hicks impossible in an uncomplicated case of placenta prævia. The first of these is where a rigid and undilated condition of the cervix makes it impossible to pass even two fingers into the uterus. The second is where the membranes have already ruptured and the uterus has so contracted down upon the fœtus that version is impossible. The third is where the presenting part is fixed in the pelvic cavity. For reasons which I shall mention later, I think the first is very much the most important. It is obvious that, if the uterine orifice will not admit two fingers, it is impossible to bring down a foot, even though we may be easily able to perform external version, and bring the breech over the brim. It is also obvious that, though this complication is rare, and, to quote an American writer, is one which "depends to a great extent upon the obstetric experience of the individual operator," it nevertheless still occasionally occurs. According to several of the speakers at the April meeting of the American Gynæcological Society, it occurs in about five per cent. of cases, and I think the Rotunda statistics would approximately agree with this figure.

It does not follow, however, because the treatment of Braxton Hicks is inapplicable in these cases, that the next best treatment is Cæsarean section, since there are at least two other methods of treatment available. It is possible to dilate or to incise the cervix to the necessary degree to enable two fingers to be passed into the uterus, and it is also possible to plug the vagina, as in accidental hæmorrhage, and so to prevent further loss of blood. Dilatation of the cervix may be dismissed, as it is too dangerous an operation in the presence of placenta prævia owing to the risk of deep lacerations of the softened cervical tissues. I have no experience, either direct or indirect, of incision of the cervix in such cases; but the criticism occurs to me that, if deep incisions of the cervix can be made with safety in the presence of a placenta prævia, as is stated by the supporters of vaginal Cæsarean section, it should be equally possible to make with safety the smaller incisions necessary to enable one to introduce two fingers into the uterus. Plugging the vagina, on the other hand, appears to be a safe and reasonable means of gaining time in which cervical dilatation can occur. It must be carried out in a careful, thorough, and aseptic manner, and under these conditions it has proved successful. If I may again refer to the Rotunda statistics, I find that among the 138 cases recorded, in four it was found

impossible to carry out the treatment of Braxton Hicks when the patient first was seen. In each of these cases the vagina was plugged, and then, as soon as dilatation was obtained, the treatment of Braxton Hicks was carried out. All the patients recovered satisfactorily, and therefore for them, at any rate, there was no need for Cæsarean section.

The second condition in which the treatment of Braxton Hicks is impossible is when version cannot be performed owing to the escape of the liquor amnii. The escape of the liquor amnii is only of importance when it is premature. If it occurs at the normal time, at the end of the first stage of labour, the patient has got to a stage at which there are plenty of satisfactory methods of treatment—if, indeed, treatment is required at all. Rupture before the second stage of labour begins is usually artificial, and is the result of a form of unjustifiable and ignorant treatment of the case. It can therefore be avoided. When it occurs spontaneously, the patient can generally be treated by plugging the vagina, as if it was a case of external accidental hæmorrhage.

The third condition in which the treatment of Braxton Hicks is impossible is where version cannot be performed because the presenting part is fixed in the pelvic cavity. Such cases, as a rule, are not serious. If the hæmorrhage persists, it can be checked by plugging if the patient is not in labour, by rupture of the membranes if she is in labour, or by extraction if the os is fully dilated. Moreover, the fact that the head is fixed would appear to contra-indicate Cæsarean section.

It is possible to conceive a case in which the vaginal plug may fail to check the hæmorrhage. We all know that under certain conditions—very rare, it is true, but still occasionally found—the vaginal plug fails to check accidental hæmorrhage because the condition of the uterine muscle is such that it relaxes before the blood-pressure and allows blood to accumulate in the uterus, even though none escapes externally. The treatment then fails, and we get a concealed hæmorrhage. Abdominal Cæsarean section followed by a supra-vaginal hysterectomy has for some time been recognized as one of the few methods of treatment of any value in the case of concealed accidental hæmorrhage, and it is most probable that in concealed unavoidable hæmorrhage similar treatment might be successful. In unavoidable hæmorrhage, however, if the leg can be brought down, the hæmorrhage will cease—whatever the condition of the uterine muscle—and, consequently, it might be possible to perform Braxton Hicks' treatment with success after a preliminary incision of a rigid and undilated cervix. Such a procedure would be of no assistance in cases of premature rupture of the membranes, but, on

the other hand, if in such cases the uterine muscle was so flabby as to allow concealed hæmorrhage, it would hardly control the fœtus to such an extent as to prevent the performance of bipolar version.

So far I have dealt with my own opinions, founded on such cases as I have treated myself and on the results of the cases treated at the Rotunda Hospital, and now I should like to turn to the opinions of others as enunciated at the April meeting of the American Gynæcological Society.

Five papers dealing with Cæsarean section in placenta prævia were read by Dr. Harrison, Dr. Jewett, Dr. Fry, Dr. Newell, and Dr. Grandin respectively, and in the discussion which followed Dr. King, Dr. Herbert Spencer, Dr. Hofmeier, Dr. Lapthorn Smith, Dr. Harris, Dr. Stone, and Dr. Peterson, in addition to others, spoke.¹ Two indications were given for the performance of Cæsarean section in the interests of the mother. The first of these was a rigid and undilatable condition of the cervix, especially in the presence of a central placenta prævia. The second was the need of some form of treatment that was suitable for use by a surgeon with an insufficient obstetrical experience. In this connexion one of the speakers (Newell) added that "the advocates of Cæsarean section had not recognized that their personal limitations furnished the great indication for an abdominal delivery, and not the exigencies of the case." The meeting as a whole was, I think, inclined to adopt the attitude that the first of these indications was present in about five per cent. of cases, and that Cæsarean section might then be justifiable. Dr. Lapthorn Smith alone seemed to consider that Cæsarean section was a comparatively safe operation in most cases, but he seemed to regard *accouchement forcé* as the only alternative. If *accouchement forcé* was really the alternative, one might be inclined to go part of the way with him, but I hope I am right in saying that in this country such treatment has long ceased to be regarded as permissible. I have tried to show why in Ireland we have not found it necessary to resort to Cæsarean section even in the five per cent. of cases in which the cervix is not sufficiently dilated to allow a foot to be drawn down, and I think everyone recognizes that throughout the British Isles the second indication is conspicuous by its absence.

I may sum up my views on the value of Cæsarean section as a means of reducing the maternal mortality in a few words. There does not appear to be any place for Cæsarean section in the modern treatment of uncomplicated cases of placenta prævia. In a few very exceptional

¹ *Trans. Amer. Gyn. Soc., Philad., 1909, xxxiv, pp. 23-72.*

cases—such, for instance, as when a rigid condition of the cervix or premature rupture of the membranes is associated with an unhealthy and atonic condition of the uterine muscle—it may be indicated. Such cases would be first treated by the vaginal plug, and, if internal hæmorrhage then results, Cæsarean section may be indicated. Personally, however, I think that if I was to meet with the unusual association of a rigid cervix with an atonic uterus—a condition which has not been seen at the Rotunda Hospital during the past twenty years—I should be inclined to enlarge the cervix by incision and then adopt the treatment of Braxton Hicks.

We must now pass on to the consideration of the foetal mortality in placenta prævia, and here we are face to face with an entirely different state of affairs. The rate of foetal mortality in placenta prævia is undoubtedly extremely high, and, furthermore, the rate apparently increases as the maternal mortality lessens. In the Rotunda Hospital, where the maternal mortality was 3·6 per cent., there were seventy-four viable infants born dead in 128 deliveries of patients with placenta prævia—a mortality of 57·8 per cent. The American statistics place the death-rate at a very similar figure (57·3 per cent.). The causes of this mortality are well known. Every factor accompanying placenta prævia tends to cause the death of the foetus. The latter is premature. Its placenta is often badly developed, is certainly in part or altogether detached, and may in addition be torn in its foetal portion so that foetal blood is lost. Consequently, since every form of treatment that gives a low maternal mortality necessitates a slow delivery, the death of the foetus is almost certain to result from asphyxia or hæmorrhage. If we want to improve the prognosis for the foetus, delivery must be effected almost as soon as the position of the placenta is diagnosed, and this, by any means except that under discussion, has been tried and has failed on account of its deadly effect on the mother. The only form of modern treatment which has apparently at all reduced the mortality of the foetus is the use of Champetier de Ribes' bag, but, as Dr. Herbert Spencer told the April meeting of the American Gynæcological Society, though it reduces the foetal mortality considerably, it increases the maternal mortality. I do not think that de Ribes' bag has been tried to any extent in Ireland, probably on this account.

Here, then, is a possible place for Cæsarean section in the modern treatment of placenta prævia. If the maternal mortality cannot be reduced, there is at least an excellent opportunity of reducing the foetal mortality. The results, however, are disappointing. Jewett, in his paper already referred to, records the results of ninety-five abdominal

Cæsarean sections with a foetal mortality of 34 per cent., of twelve utero-vaginal sections recorded by Bumm with a foetal mortality of 83·3 per cent., and of twenty-six vaginal sections recorded by Hammerschlag with a foetal mortality of 55 per cent. These figures show that vaginal Cæsarean section as a means of improving the foetal mortality need not be considered, and that abdominal Cæsarean section alone is of any avail. The latter, according to Jewett's figures, effects a reduction of about two-fifths, and, presumably, if it was performed at an earlier stage in the case, it would be more successful. It is, however, obvious that, if Cæsarean section is to be of use, it must be adopted in all cases, since we have no means of telling when the life of the foetus is in danger, and when it is not; and it must be adopted to the exclusion of every other mode of treatment, otherwise it is probable that the foetus will die during the delay thus caused, and a subsequent Cæsarean section be valueless. Seeing that, after Cæsarean section, the maternal death-rate is nearly four times greater than after the usual obstetrical methods, and that the prospects of the life of the foetus are bad, owing to its prematurity and the effects of the previous hæmorrhages, I do not think anyone is prepared to recommend such a course. Consequently, Cæsarean section is only likely to be performed for the sake of a foetus whose life is of exceptional importance.

Before I finish, I ought perhaps to say a few words on the relative advantages of abdominal and vaginal Cæsarean section respectively in the few cases in which either is indicated in the interests of the mother alone. If one regarded Cæsarean section as an advisable routine treatment in the mother's interests for placenta prævia, one might also be inclined to regard the vaginal operation as preferable to the abdominal, provided it could be shown to be as safe. If, however, one considers that Cæsarean section should be reserved for cases in which a rigid condition of the cervix is associated with an atonic condition of the uterine muscle, one must take the possibility of severe *post-partum* hæmorrhage into account, and this may materially modify the situation. If the uterus does not contract after an abdominal Cæsarean section, supravaginal hysterectomy is a comparatively short and simple procedure, while the removal of the full-term or nearly full-term uterus through the vagina is a longer and more complicated procedure. The very fact that vaginal Cæsarean section has been successfully practised in placenta prævia seems, if I may say so, to prove that it is never required, since, if one can make the deep incisions necessary to remove the foetus, one ought to be able with still greater ease to make the smaller incisions necessary to enable us to perform bipolar version.

Lastly, so far as the foetus is concerned, it is apparent that the statistics collected by Jewett are almost as fatal to the vaginal operation as the operation is to the foetus.

In conclusion, I have only to say that I hold no brief against the adoption of Cæsarean section as a treatment for placenta prævia. My position is simply that I am unable to see a place for it. What we really require in these cases is an advance in our methods of treating the collapse of hæmorrhage and the shock, and of combating infection, as thus we could reduce the present mortality and save some of the patients who are apparently moribund when they first come under treatment. In all other cases, our existing methods appear to me to be sufficient so far as the mother is concerned.

DISCUSSION.

Dr. HERBERT SPENCER congratulated Dr. Jellett on his excellent paper, which was particularly valuable at the present time when Cæsarean section was being extensively advocated for conditions which permitted natural delivery in many cases, and in others permitted delivery by methods much less serious to the mother than Cæsarean section. Thus a foreign gynæcologist had recently indignantly repudiated the suggestion that some of his Cæsarean sections were unnecessary by asserting that he had only performed Cæsarean section 40 times out of 2,500 labours, or once in 63 labours! Dr. Jellett had done well to show the small mortality that attended the treatment by bipolar version and slow delivery; the cases which terminated fatally did so as a result of severe hæmorrhage or infection before the treatment was adopted, and would not be saved, but would be further imperilled by the performance of Cæsarean section, unless, in the infected cases, the uterus was removed. He (Dr. Spencer) did not go so far as Dr. Jellett, who said there was no place for Cæsarean section in the treatment of placenta prævia, but thought it was clearly indicated in certain rare cases of complete placenta prævia at or near term with a living child, an undilated rigid cervix, and a considerable loss of blood. In such a case the delivery by the natural passage was extremely dangerous to the mother and almost certainly fatal to the child, whereas Cæsarean section would almost certainly save the lives of both. At the annual meeting of the American Gynæcological Society, held in New York last year, he had mentioned such a case of his own which occurred over twenty years ago, and which terminated fatally as a result of hæmorrhage and shock from dilating the cervix and delivering by the vagina.¹ It had always been a regret to him that he had not performed Cæsarean section in that case. But it was the only case he could remember in which he thought Cæsarean section

¹ *Trans. Amer. Gyn. Soc., Philad., 1909, xxxiv, p. 65.*

was called for, and amongst the many distinguished American obstetricians present at the meeting alluded to there was as much unanimity as is ever met with amongst medical men that the operation was called for only in the rarest instances. If, however, the interests of the child are to be placed on the same level as the interests of the mother (a position which he thought no one could defend), then de Ribes' bag and Bossi's dilator greatly improved the chances of the child surviving without greatly endangering the mother.

He drew attention to the remarkable series of cases of placenta prævia treated by Cæsarean section by Krönig¹ and Sellheim, who had performed the operation twenty-six times, all the mothers recovering, all the children being born alive, and all who weighed over two kilos surviving. These remarkable results should arrest the attention of all obstetricians; but, until the cases had been published in detail, he did not feel inclined to alter his opinion that placenta prævia very rarely indeed called for Cæsarean section, an operation which could not be safely performed at the patient's home, which required time, aseptic surroundings and skilful assistance, and which left behind it several potential disabilities: whereas version could be safely carried out in a few minutes without assistance, and, if followed by slow delivery, interfered in no way with the patient's well-being, nor with subsequent parturition.

Dr. CHAMPNEYS expressed his appreciation of the lucid and logical paper which they had just heard, and with which he almost entirely agreed. He thought that in placenta prævia the child must take its chance; that the mother must be the first consideration, and that any special attempt to improve the chances of the child would be at the expense of the mother. Secondly, he thought it was satisfactory to find in any obstetric question that the best treatment could be carried out by Nature's weapons—in this instance Braxton Hicks's bipolar version, which, invented many years ago, had become overlaid, was rediscovered in Germany some twenty-five years ago, and now was the accepted routine treatment in the Rotunda. It was occasionally possible to perform it through a cervix which only admitted one finger; he had done it himself.

Dr. PURSLOW said that he believed that good results could be obtained in those cases for which Cæsarean section was proposed—viz., where the os would not admit of bipolar version; by the older treatment of plugging, but, unless the vagina was tightly packed with the material, the proceeding was worse than useless, and, in order to secure tight packing, it was essential that the perineum and posterior vaginal wall should be well retracted: if possible, a Sim's speculum should be used for this purpose, and it was a great advantage to have the woman in the dorsal position. In his experience *post-partum* hæmorrhage was a complication greatly to be dreaded in cases of placenta prævia, and he believed that it might occur independently of lacerations of the cervix, owing to the fact that the placental site was in an unfavourable position to allow of hæmostasis by uterine contraction; although the amount of blood

¹ *Berl. med. Wochenschr.* 1910, xlvii, p. 7.

lost might be small, yet, coming on the top of the previous hæmorrhage, it might quickly place the woman in a condition of grave danger. Cæsarean section would not provide against this contingency, unless the uterus were removed at the same time.

Dr. AMAND ROUTH agreed with Dr. Jellett that there was no place for Cæsarean section in the ordinary or routine treatment of placenta prævia. An American textbook recently published suggests that Cæsarean section might be done when the *ante-partum* hæmorrhage was uncontrolled by a tampon, where the os is undilated, the woman not much affected by the hæmorrhage, the child at term and living, and the patient uninfected and with a good environment. This would mean that Cæsarean section would become the general treatment of cases of placenta prævia seen in their early stages, and would be undertaken before a sure diagnosis was made, and before dilatation of the cervix and Braxton Hicks's method of version had been attempted. This is clearly inadmissible and unjustifiable, but it is quite possible that it would be good treatment in some of the cases described by the author where the cervix was rigid and undilatable. According to the author this condition occurs in 4 or 5 per cent. of all cases. He recalled such a case more than twenty years ago, where serious *ante-partum* hæmorrhage had taken place in a primipara at a seaside resort. When he reached the patient he found the vagina plugged by the doctor in charge of the case, the external os was closed, and only with the greatest difficulty—after two hours under deep anæsthesia—could he get two fingers in, and eventually was able to bring down a foot and arrest the hæmorrhage. Next morning the cervix was still absolutely rigid, and did not yield for another fifteen hours, when the child was born. The cervix was, however, torn, and the patient eventually died of sepsis. If he had that case to-day, with the added twenty years' experience, he would certainly do Cæsarean section. If Cæsarean section were reserved for such cases of placenta prævia it would not be adding the 8 per cent. risk of Cæsarean section to the 8 per cent. risk of placenta prævia, but would merely substitute the one for the other, adding therefore no extra risk. He did not think that incisions of the cervix in such cases of rigid cervix would be desirable, for such incisions would certainly become extended into the lower uterine zone as the after-coming head was being born. In cases of placenta prævia treated by Cæsarean section, he thought the incision should be a fundal one to avoid encroaching on the placental site, for one of the dangers of Cæsarean section in these cases was that of *post-partum* hæmorrhage afterwards.

Dr. W. J. GOW considered that in discussing the place of Cæsarean section in the treatment of placenta prævia it was important to remember that there were different varieties of this condition, and that in central placenta prævia alone treatment by Cæsarean section deserved consideration as a method of treatment. In many cases of lateral and marginal placenta prævia delivery by version presented no particular difficulties or dangers, whereas in central

placenta prævia the mother's life was often exposed to serious risk, and the child seldom or never survived. He mentioned that he had performed Cæsarean section for one case of central placenta prævia in which both mother and child did well. Such treatment was only to be undertaken in exceptional cases. He considered that the indications for such a form of treatment were as follows ; (1) That the placenta be centrally situated ; (2) that the os be small and rigid ; (3) that the parts bleed freely on any attempt at manipulation ; (4) that the surrounding conditions be favourable ; (5) that the mother be not in a condition of collapse ; (6) that the child be near full term and alive. In the hands of a skilled operator the proceeding presents no difficulties, and very little danger to the mother. The life of the child is saved, whereas by other methods of treatment it would almost certainly be lost. The treatment by hydrostatic bags or bipolar version is associated in such cases with a very real danger to the mother, and the risk attending a Cæsarean section is certainly not greater, and probably considerably less. Previous speakers had alluded to the risk of *post-partum* hæmorrhage after delivery by version in cases of placenta prævia. He thought it probable that in such cases the bleeding came from lacerations, produced during extraction, rather than from an imperfectly retracted lower uterine segment. In the case in which he had performed Cæsarean section he was struck by the anæmic condition of the uterus, and the firm contraction which took place after the child and placenta were removed. Another class of case in which he also thought Cæsarean section was indicated was where the patient, in addition to a central placenta prævia, had a purulent vaginal discharge. The risk of infection in these cases was very great, inasmuch as the fingers passed through the vagina had to be brought in direct contact with the placental site. Cases of purulent *ante-partum* vaginal discharge were common at Queen Charlotte's Hospital, probably gonorrhœal in origin, and as long as the third stage of labour was normal, no trouble occurred during the puerperium ; but if the placenta had to be removed, unusually grave septic infection frequently followed.

Dr. GRIFFITH had so recently referred to his one case of Cæsarean section for placenta prævia in the discussions at that section, and on Dr. Blacker's paper at the Harveian Society,¹ that he would not occupy the time of the Section with any detail. It seemed to him altogether monstrous to talk of Cæsarean section as the treatment for placenta prævia, except in very rare and exceptional cases. So many cases occurred in multiparæ in which the patient could be safely delivered by the methods generally accepted, and it was only in cases of great difficulty and danger to mother and child that the operation should be recommended.

The PRESIDENT (Dr. Macnaughton-Jones) expressed the indebtedness of the Section for the valuable paper which Dr. Jellett had read. He had utilized the experience of a historic hospital, which, he was safe in saying, had attracted, and still did attract, more students from distant parts to study midwifery than

¹ *Lancet*, 1910, i, p. 793.

any other existing institution of a similar character. He (the President) still believed in the soundness of the principles which he had taught students in the past to follow in the management of the different forms of placenta prævia; the two most important of these being the indications for plugging and bipolar version. Robert Barnes, whose name must ever be associated along with that of his colleague, Braxton Hicks, in the modern treatment of placenta prævia, as far back as 1864 published the results of sixty-nine cases, in which there were six deaths. Two of these were moribund when admitted to hospital, and all were of a bad type. He then said that he felt sure that if we could always see these cases at the earliest stage of the hæmorrhage, and if they were treated on correct principles, the mortality would be reduced to a point not hitherto known. Perhaps the most complete résumé of the entire subject hitherto published was that of Ludovico Moncalvi (1909), assistant to Professor Mangiagalli, in the Institute of Obstetrics and Gynæcology in Milan. In that institution during the years 1907, 1908, 1909, out of 1,821 deliveries there were 45 cases of placenta prævia, with a mortality of 6 of the latter—22 of these were central, 16 marginal, and 17 lateral. In 4 of the 6 fatal cases the attachment was central, and in none of them was it possible to adopt Braxton Hicks's treatment; though it is evident from the statistics that it is largely adopted in the hospital. Sepsis and death followed the one case of central attachment in which Cæsarean section was performed. Moncalvi's monograph was well worth perusal, as it entered fully into the relative importance of such matters as the nature of the placental presentation, the size and weight of the fœtus, the character of the presentation, whether vertex or otherwise, the causes of death, giving the statistical records on all these points of several of the largest clinics of Europe. The whole weight of the evidence is on the side of those who maintain that only in a very limited number of cases, if the condition be treated on scientific obstetrical principles, is Cæsarean section called for or justified. Professor Pinard, of Paris, wrote to him (the President) that from 1898 to 1908 there were 183 cases of placenta prævia in the Baudelocque Clinic with four deaths, a mortality of 2·18 per cent. A Porro's operation was successfully performed once in a case of pelvic deformity, but no Cæsarean section. Compare this with Professor Krönig's practice from the statistics he had kindly sent to him from the Freiburg Clinic: Forty cases of placenta prævia, total mortality one, 2½ per cent.; twenty Cæsarean sections were performed without a death. It must be remembered that these results were achieved by an exceptionally brilliant and experienced surgeon in one of the most perfectly equipped clinics. Dr. Jellett had been quite right to exclude from the scope of the discussion all those cases in which, even if the placenta were attached in the normal situation, Cæsarean section might be indicated. Statistics to be of real value, from which to draw any line of practice, should specify the exact indications for operation, also the several accessory conditions which attended on the placental presentation, such as that rightly insisted on by Dr. Gow, whether it were central or marginal; the presentation, position and size of the fœtus, the stage of the labour and the state of the membranes;

whether in a primipara or multipara, and if any deformity of the pelvis existed. For example, in Professor Krönig's twenty cases he would wish to have these particulars. It seemed to him (the President) that this was a proposal to take the treatment out of the hands of the skilled obstetrician and place it in those of the skilled surgeon, it being taken for granted that the resources of the obstetric art were exhausted. The statistics quoted at the American meeting by Fry and others, and the views held by American obstetricians generally, endorsed by more recent writers, showing the relative results to the mother and child from Cæsarean section and those that followed other methods of treatment, confirmed him in the view that so far the evidence was altogether on the side of those who resorted to operation only under very exceptional circumstances, and in which it was not possible to practise such means as those referred to by Dr. Jellett.¹

Dr. HENRY JELLETT, in reply, thanked the Fellows and Members of the Section for the extremely kind manner in which they had received him and had criticized his paper. When he wrote it, he thought that perhaps he might be expected to recommend Cæsarean section in these cases, and he was now glad to find that the Section as a whole agreed with his condemnation of it, except in the rarest cases. His views on the subject were based on the statistics he had laid before the Section, and he did not see how anyone could hold different views unless he was prepared to say that he did not regard the Rotunda cases as typical. Dr. Herbert Spencer considered that an occasional case of placenta prævia, in which Cæsarean section was required, might occur, and of course one could not say that this might not be so. Still, the Rotunda statistics were based on a very large number of cases, and in no instance was it apparent that Cæsarean section would have done any good. Dr. Spencer had also alluded to Krönig's twenty-six successful cases of Cæsarean section without any deaths, but this was probably only an accidental series of picked cases. It must be remembered that when Veit introduced the morphia treatment of eclampsia, and Bumm its treatment by immediate delivery, they both got a long series of cases without mortality, and this success subsequently was not maintained. He (Dr. Jellett) quite agreed with Dr. Champneys, in thinking that one must consider the life of the mother in cases of placenta prævia, and

¹ The following statistics did not arrive in time to utilize in the discussion:—Dr. Thrès, from Professor Bumm's clinic in Berlin, records from the year 1904 to 1909 179 cases of placenta prævia. The total maternal mortality was 5 (2·85 per cent.), 94 of the foetus (52 per cent.) Fifteen vaginal Cæsarean sections were performed, with one maternal death (6·6 per cent.); mortality of the foetus, 7 (46·6 per cent.). Professor Dimitri de Ott, from the Imperial Institute of Obstetrics and Gynæcology, St. Petersburg, writes: During eighteen years there were 111 cases of placenta prævia, with a total maternal mortality of 2·51 per cent., and of the foetus 71 per cent. No Cæsarean section has been performed. From Sir Halliday Croom I learn that no Cæsarean section for placenta prævia has been performed to his knowledge in Edinburgh, and Dr. Munro Kerr (Glasgow Maternity) says that the only case in which he performed Cæsarean section for placenta prævia was the successful one already reported. He is "convinced that the operation has a place in certain well-chosen cases, but these are very few and far between."

that the fœtus must take its chance, as experience had taught that the two were, so to speak, antagonistic, and that efforts to save the child only caused a higher maternal mortality. He quite concurred with Dr. Purslow, in thinking that it was impossible to plug the vagina satisfactorily without a speculum in these cases. *Post-partum* hæmorrhage was a real danger after placenta prævia, and it was a golden rule, if it occurred, immediately to plug the uterus without waiting to try other methods, as by so doing any further loss of blood was prevented. In answer to Dr. Routh, Dr. Jellett said that he believed the maternal mortality in a long series of cases of Cæsarean section was about four times greater than the mortality after Braxton Hicks's treatment. Dr. Gow said that we should discuss central placenta prævia alone, as it was only in case of it that Cæsarean section was required. Of course central placenta prævia is more serious than marginal or lateral, but that the latter might also be serious was shown by the Rotunda statistics, as, amongst the five fatal cases, there were three cases of marginal and one of central placenta prævia. He did not agree with Dr. Gow that *post-partum* hæmorrhage was always necessarily due to laceration of the cervix. He thought Dr. Griffith's case was exceptional, and came under the head of those in which the life of the fœtus possessed special importance. Under such circumstances, Cæsarean section in the fœtal interests might undoubtedly be justifiable. He thanked the President for his remarks, and hoped that, in the present case, the statistics presented were reliable, and that he had escaped the dangers which surround all collectors of statistics.

A very large Cervix Fibroid.

By A. E. GILES, M.D.

DR. ARTHUR GILES said that the specimen was interesting from two points of view, that of diagnosis and that of the origin of the tumour. The patient, aged 50, was admitted to the Prince of Wales's General Hospital, Tottenham, on February 8, 1910. She had had seven children, the youngest aged 9, and one miscarriage seven years ago. For a year after the miscarriage, menstruation was profuse and ceased altogether at the age of 44; thereafter her health was apparently good until a year and a half ago, when she noticed some abdominal swelling; lately this had increased considerably. For the last year there had been frequency and sometimes difficulty with micturition; she had had no pain at all except at times a little on passing water; the bowels were rather constipated. On admission there was a large irregular tumour reaching up to the ensiform cartilage and giving the impression of fluctuation; the cervix was small and drawn high up behind the pubes. The patient looked ill and emaciated, the appearance being suggestive of the cachexia of malignancy. A diagnosis of ovarian tumour was made, and it was thought that the tumour was partly solid and perhaps malignant. On opening the abdomen it was seen that both ovaries were normal and independent of the tumour; the latter occupied principally the position of the right broad ligament, the peritoneum of which was enormously stretched. Such difficulty as the operation presented was due principally to the distortion of normal relations and the difficulty in finding the uterine arteries. A forceps was placed on the right ovarian artery, the broad ligament was opened by a long incision in front and behind, and the tumour shelled out; it was then seen that it was connected with the uterus, and when the tumour had been raised right up, the uterine arteries were secured; the edges of the broad ligament were brought together by a continuous suture, the line of suture from the stump to the right ovarian artery being about 12 in. long. The patient made an uninterrupted recovery and left the Hospital on the twenty-sixth day. The tumour weighed 23½ lb., and investigation showed that it originated from the posterior wall of the cervix.

It was very unusual for cervix fibroids to attain any great size, as their development was generally limited by the dimensions of the true pelvis. As far as he had been able to ascertain, this was the largest tumour of the kind that had been reported.

Report of the Pathology Committee.—"We have examined the specimen exhibited by Dr. Giles of large cervical fibroid, and are of opinion that that description is correct. The fibroid has originated from the right posterior aspect of the cervix, and, developing thence into the right broad ligament, has elevated the peritoneum greatly above it. The sections show cystic and hyaline degeneration of a uterine fibroid."

DISCUSSION.

Dr. PURSLOW said that he had removed a very similar specimen last year, and had recorded the case in the *Birmingham Medical Review* for October, 1909¹; the specimen was now in the University Museum at Birmingham. The tumour grew from the anterior wall of the cervix and its attachment was clearly seen, as a sagittal, mesial section of uterus and tumour was made after removal. The uterus was of normal size; the whole mass weighed 20½ lb. A remarkable feature was that the patient made no complaint beyond that of dyspnoea and a feeling of weight in the abdomen, and was getting about until her admission to Hospital. He closed the broad ligament from above, after putting a gauze drain into the posterior fornix, and the patient did well.

The PRESIDENT said that he had recorded a case in which he removed a giant myoma weighing 28½ lb. The enormous solid tumour, which measured 16 in. in its long diameter, had a dual origin. It sprang by one root from the broad ligament, and by another from the uterine wall above the cervix. He opened the bladder in its removal, but this was closed at the moment, and the patient made a complete and permanent recovery.

Dr. FLORENCE WILLEY said she had removed a similar tumour, weighing 16½ lb., from a patient, who was small in stature so that the tumour reached beneath the costal margin. It was thought before operation to be ovarian in origin. There were no menstrual or other symptoms, and the patient's only complaint was a growing sense of weight. It was found to be an intraligamentous fibroid attached to the lower uterine segment.

¹ *Birm. Med. Rev.*, 1909, N.S., xiv, p. 169.

Sarcoma of the Body of the Uterus from a Girl aged 20.

By HENRY JELLETT, M.D.

THE specimen had been removed by Dr. Jellett two months ago. The growth is very clearly to be seen and both involves the mucous membrane and also occurs as nodules in the uterine wall. Further, there is a large nodule in the right broad ligament lying between the uterus and the ovary and attached to both. This nodule is also sarcomatous in nature. The patient complained of menorrhagia and constant purulent discharge from the vagina, and on examining her before the operation a quantity of pus escaped from the uterus. Dr. Jellett thought the case was one of myoma, and so he removed the uterus supra-vaginally, removing also the entire broad ligament and ovary on the right side. On receiving the report of the pathologist, Professor O'Sullivan, on the nature of the case, he at first intended to remove the remainder of the cervix, but as it was definitely separated from the growth in the uterus, and as Professor O'Sullivan did not consider its removal necessary, he allowed it to remain. The patient made an uninterrupted recovery, and, so far, shows no sign of any return.

Professor O'Sullivan's report on the tumour is as follows: "The uterus was large, the body being 4 in. in length, and the wall a little over 1 in. thick at the thickest part. The upper two-thirds of the cavity was filled by an irregular growth with numerous polypoid excrescences projecting into the lumen. The surface of the growth was acutely inflamed and partially necrosed, and covered in places with a thick layer of pus which contained a variety of organisms, bacilli, and cocci. The growth could be seen to infiltrate the posterior wall of the uterus, and secondary nodules could be felt in the upper and posterior part of the wall. The ligament of the ovary was also infiltrated, and the ovary itself was converted into a mass, partly nodular and partly cystic. Under the microscope the tumour showed a very cellular structure, spindle and large round cells with numerous nuclear figures. The solid tumour in connexion with the ovary had a similar structure. A large nodule lay in the upper uterine wall, which was of a dull grey colour on section, and proved to be of similar structure."

Report of the Pathology Committee.—"We have also examined sections of Dr. Jellett's case of sarcoma of the uterus, and are of opinion that its general characters are those of sarcoma. Some of the details of the sections suggest an endothelial origin—namely, the alveolated character of the growth, the absence of stroma in places, and the well-defined blood vessels."

Specimen of Torsion of the Body of the Uterus.

By W. S. A. GRIFFITH, M.D.

THE patient, aged 67, was under the care of Dr. Crossley-Wright, Halifax; married, no pregnancy. Menstruation from the age of 14 to 42. No menorrhagia, but usually severe pain. From the age of 25 she was under the care of the late Sir Spencer Wells, who diagnosed a fibroid and sent her to Woodhall Spa, where she obtained so much benefit that the tumour apparently ceased to grow, and gave her little trouble, she remaining under his observation. From the time of the climacteric until two years ago she had no trouble whatever, but during the last two years she had had four attacks of very severe abdominal pain, each attack beginning on rising from bed in the morning, accompanied by profuse sweating and vomiting; the last attack occurred a few days before she was seen by Dr. Griffith on March 8. For some years the bowels had been acting only with increasing difficulty and under large doses of medicine, and the tumour had been apparently increasing in size. The tumour was found to extend from the brim of the pelvis to the upper border of the navel, about the size and shape of a pregnancy at the end of the fifth month. It was dense, with a feeling of elasticity, and rather sensitive to pressure. At the operation on March 11 the uterus and tumour were found twisted to the right, the left broad ligament, the veins of which were much dilated and engorged, being on the right and the posterior surface of the uterus in front. Supravaginal amputation was performed after the omentum had been separated from the brim of the pelvis and the fundus. There was no free fluid nor other evidence of peritonitis. The patient made an uneventful recovery, and returned home within a month of the operation.

The tumour was a fibromyoma, undergoing mucoid degeneration.

The PRESIDENT (Dr. Macnaughton-Jones) said that some years since Ehrendorfer had accounted for the twisting of the tumour by the resistance offered to its growth from the infringements on the pelvic wall, or from the presence of an ovarian tumour. The consequences were often serious, as it led to fibromitis, necrobiosis of the tumour, and peritonitis; it was also the source of considerable pain.

Large Cervical Fibroid in the Vagina obstructing Labour.

By W. S. A. GRIFFITH, M.D.

THE patient, aged 39, was admitted under Dr. Griffith's care, labour having begun the day before. There was nothing important in her previous history, her four previous labours being natural. For two months before admission she had complained of a thick, yellow discharge. A week before admission, while walking, she first felt a lump pressing at the vulva. On examination under an anæsthetic a purplish œdematous mass was found distending the vagina and sloughing at its lowest part. With difficulty the hand was passed in front of it and the dilated cervix with the foetal head presenting; the tumour, attached to the whole breadth of the posterior cervical wall, was drawn down and removed by enucleation and division of the pedicle. The blood-vessels and the capsules were large, and bled profusely; the bleeding, though controlled by pressure, was not arrested until the child was delivered with forceps and the uterus contracted down. The patient and child have made an uneventful recovery.

Examined this day, April 14, a fortnight after delivery: The involution of the uterus is normal; the sac of the fibroid only admits a finger. The tumour weighed over 2 lb., and was 15 in. in circumference; is a fibromyoma undergoing mucoid degeneration,

A Fibroid of the Cervix and Body of the Uterus.

By J. P. HEDLEY, M.C.

THE specimen is a uterus with a single large fibromyoma in its wall, and is somewhat remarkable in that the tumour occupies the entire posterior wall of both cervix and body, expanding these two parts of the uterus so evenly that it is impossible to tell where the cervix ends and the body begins. The cavity of the uterus is laid open anteriorly, and the tumour is cut almost completely into right and left halves from behind; on the cut surfaces several large blood-vessels are seen. The lower part of the tumour is uncovered by peritoneum. The fibroid is egg-shaped, the larger end being at the fundus; it measures 9 in. in length, and 4 in. in greatest breadth; the uterine cavity is 8 in. long, but its breadth is only slightly increased, except in its lower part. The specimen weighs $6\frac{3}{4}$ lb. Microscopical examination shows the tumour to be a fibromyoma, with an unusually large proportion of fibrous tissue in it.

The patient from whom the specimen was removed was a woman aged 30, who had been married for nine years and had had no pregnancy. Menstruation began when she was aged 14, and was regular and not excessive; for two years she had noticed a progressive increase in the amount lost, and that the duration of the periods gradually lengthened from four to eight days. For one year she had noticed an increasing swelling in the lower part of the abdomen, and there had been frequency of micturition from time to time. On examination the tumour was found to extend to a point $2\frac{1}{2}$ in. above the umbilicus, and presented the ordinary features of a fibroid of uterus. *Per vaginam* the anterior lip of the cervix was found to be thin and stretched; the posterior much enlarged and the os crescent-shaped.

The operation was performed by Dr. Tate on April 7 of this year; there was considerable difficulty in shelling out the lower part of the tumour, especially on the right side, but there was comparatively little bleeding, and the patient stood the operation well. She is now progressing favourably.

**Pregnancy in a Septate Uterus treated by Abdominal
Hysterectomy Five Months after the Death of Fœtus
at Full Term.**

By WALTER TATE, M.D.

THE patient from whom the specimen was removed was a married woman, aged 29. She had had three children, the youngest having been born four years ago. Since then she had menstruated regularly and had had good health up till January, 1909, when she had her last normal period. From this date the various signs of pregnancy gradually developed—morning sickness, enlargement of the breasts, and increasing size of the abdomen, and the patient was quite satisfied that she was pregnant. In October—that is, after nine months of amenorrhœa—she had an attack of abdominal pain which lasted for one week, and then passed off. A midwife was sent for, as the patient thought she was in labour. After this attack of pain the abdominal enlargement ceased to grow, but the pains have recurred at intervals, sometimes coming on every day for a fortnight. They start as soon as the patient gets up in the morning, and last about fifteen minutes. In January she went to a doctor, who told her she was several months pregnant. As, however, she was not satisfied, she consulted another, who was of opinion that there was a fibroid tumour of the uterus. In January of this year some hæmorrhagic discharge began with the passage of a few small clots. It had occurred nearly every day up to the time of her admission to St. Thomas's Hospital, on March 21, 1910.

The patient appeared to be a strong, healthy woman. On examining the abdomen there was a very obvious, prominent swelling, about the size of a seven months' gestation. The tumour was symmetrical in shape, and was of firm consistence, with a sense of elasticity in parts. It was fairly mobile, and presented the characters of a uterine fibroid, except that over a small area below and a little to the right of the umbilicus a sensation of crackling could be felt, suggesting the presence of a dead fœtus. The fœtal parts could not be defined. *Per vaginam* the cervix was small, and showed no changes suggestive of pregnancy. Through the anterior fornix a firm mass could be felt continuous with

the cervix, and the whole abdominal tumour seemed to be a direct expansion from the cervix. The sound can be passed for a distance of $2\frac{1}{2}$ in. in a backward direction.

Abdominal section: A median incision was made from 1 in. above the umbilicus to the symphysis. On exposing the surface of the tumour it had the appearance of a large uterine fibroid. Over the posterior and upper surface of the tumour the omentum was adherent. After separating this, the posterior surface of the tumour had a grey, necrotic-looking appearance. The Fallopian tube and ovary were seen on either side of the tumour, on the right side at a much higher level than on the left. The foetus could now be easily felt through the wall of the tumour, and at this stage of the operation the pregnancy appeared to be a uterine one. The whole tumour was now brought outside the abdomen, and abdominal hysterectomy was performed without any difficulty. The right appendages were removed with the tumour. The left ovary and tube were normal, and were not removed. The vagina was left open, and the peritoneum closed over it in the usual manner. The abdominal wound was closed in layers. The patient made a rapid recovery. She was allowed to get up on the fourteenth day after her operation, and was discharged on the seventeenth day.

Parts removed: The tumour removed is about the size of a seven months' pregnancy, and appears to be the uterus enlarged by a tumour. The right side of the mass reaches a higher level than the left, and right uterine appendages spring from the mass at a higher level than those on the left side. The anterior surface is everywhere smooth, the posterior surface is roughened over a large area, where the omentum was adherent.

On laying open the uterine canal, which was $3\frac{1}{2}$ in. long, it was found to extend upwards and backwards at the posterior and left aspect of the whole tumour. An incision made over the anterior surface of the tumour in the right side showed a muscular wall about $\frac{1}{5}$ in. to $\frac{2}{5}$ in. in thickness, covering the foetal membranes and thin edge of placenta. On tearing through the membranes, an ounce or two of thick, brownish amniotic fluid mixed with meconium escaped, and the back and shoulders of the foetus presented. An incision on the posterior necrotic surface of the tumour showed the wall here to be very thin, less than $\frac{1}{8}$ in. in thickness. A small window through the cavity of the uterus showed a muscular septum of $\frac{1}{4}$ in. in thickness, separating the uterine cavity from the sac containing the foetus. There is no sign of any communication between the cervical canal and the gestation sac.

Report of the Pathology Committee.—“We have examined the specimen with microscopic sections exhibited by Dr. Tate, under the title of ‘Doubtful Interstitial Pregnancy,’ and are of opinion that it is a pregnancy in one half of a septate body with a single cervix. No communication between the cervix and the pregnant side of the uterus can be demonstrated. The inner lining of the gestation sac is too necrotic to demonstrate whether a tubal or endometrical lining exist.”

Dr. MAXWELL referred to a similar case shown several years ago by Dr. Andrews. In that case a small unimpregnated uterus lay alongside of, and was firmly incorporated with, a large irregular cavity which had no connexion with the single cervix. The larger tumour contained several pints of foul, infected fluid (*Bacillus coli*) and a macerated, well-developed, full-time foetus. Investigation of the case showed this cavity to be the right half of a uterus duplex containing a foetus that must have entered it by the process of “external migration.” So far as a superficial examination of the specimen shown that night entitled one to express an opinion, Dr. Maxwell thought the case was more probably of this nature than an interstitial pregnancy. The relations of the three structures—ovarian ligament, tube and round ligament—were normal, and it was difficult to conceive an “interstitial” pregnancy without considerable distortion of the round ligament. The length of gestation, too, was a point that favoured development of an ovum in one of the cavities of a duplex uterus rather than in an “interstitial” site, and, similarly to Dr. Andrews’s case, the ovum must have entered by “external migration.”

Obstetrical and Gynæcological Section.

May 26, 1910.

Dr. H. MACNAUGHTON-JONES, President of the Section, in the Chair.

Cæsarean Section in the Treatment of Eclampsia Gravidarum, with Notes of a Successful Case.

By FREDERICK MCCANN, F.R.C.S.

THE treatment of puerperal eclampsia is a subject of the highest importance to all those engaged in the practice of obstetrics, and is one in which there is opportunity for considerable difference of opinion. This diversity of opinion, which has existed for many years, and still exists, is due mainly to the ever-changing views regarding the pathology of the disease. At present opinions as to treatment may be divided into two groups:—

- (1) Those favouring expectant treatment by drugs and other means.
- (2) Those favouring immediate and rapid delivery.

In the *Journal of Obstetrics and Gynæcology of the British Empire* for 1904, v, p. 263, Comyns Berkeley has collected the opinions of a number of obstetricians as to how they would treat puerperal eclampsia. The extraordinary divergence in the methods recommended is the most striking feature in this essay. Certain of those who have replied to the questions asked recommend morphia, because it hinders metabolism, whilst others recommend thyroid extract because it aids the metabolism of nitrogenous substances. Even in the use of purgatives there is absence of unanimity—some strongly advocate their use, others think they are harmful. There seems to be more agreement concerning abdominal and vaginal Cæsarean section, for both operations are regarded by the majority as being unjustifiable.

There are few who have had a sufficiently large experience of this disease to enable them to dogmatize over the methods of treatment. Indeed, the more cases of eclampsia one sees, the more is one impressed with the great variability in the symptoms, course, and sequel of this disease. There is further considerable difficulty in appraising the true value of many of the methods of treatment, for from 75 to 80 per cent. of the patients recover, and our methods are as yet purely empirical, for we do not know the cause or causes of the disease. The toxic theory which now finds favour is nothing more than a return to the humoral pathology of our forefathers, and the treatment advocated in bygone days, founded on that pathology, compares favourably with the so-called modern methods.

Unfortunately, under the term "puerperal eclampsia" more than one disease is included, for some writers seem to have grouped under this designation all those conditions in which fits occurred, and for this reason many statistical tables have to be disregarded.

I was led to consider anew the question of rapid delivery in this disease through having encountered in my private work a severe example of eclampsia gravidarum.

A lady, aged 26, a primipara, had been married for a year and had always enjoyed good health. She missed her first period in September, 1907, and was examined by her medical attendant when about six to eight weeks pregnant. At that time she was in good health, and everything was reported to be normal. She went to the South of France and remained until February, 1908. On her return she was again seen by her doctor, when she expressed herself as feeling very well indeed, but unable to walk as far as she had been in the habit of walking. Early in April her father became seriously ill, and ultimately died on April 30. She was very much attached to her father, and his illness came as a great shock to her and completely upset her nervous system. On April 19 her own doctor was sent for, and he found her complaining of indigestion, flatulence, and other signs of gastric disturbance. On April 20, 1908, she was not feeling very well all day, but was able to be up and about the house, although complaining of headache. Suddenly at about 8 p.m., without any warning, she had two or three very bad fits. I was telephoned for and told that in the last fit she had nearly died, and her condition was so bad that the case was hopeless. An injection of morphia was given. I reached the house about 10 p.m., and found the patient in a semi-comatose condition. Her face was livid and her eyelids swollen. A small

quantity of urine was withdrawn by catheter and found to be solid with albumin. She had another fit in my presence. Her pulse was very rapid, and her tongue had been badly bitten. I at once decided to empty the uterus, and for this purpose she was put deeply under chloroform, and an attempt made to dilate the cervix—first with metal dilators and subsequently with the gloved fingers. The cervix was elongated and extremely rigid; indeed, I had never encountered such a degree of rigidity. As little or no progress was being made with the dilatation, and as the fits were constantly recurring, and of such severity that on more than one occasion she appeared to be dying, I decided that the best course to pursue was to empty the uterus by the Cæsarean operation. After having explained the situation to her husband and obtained his consent, a rapid Cæsarean section was done at 2.30 a.m. (April 21). Whilst undergoing the operation the patient had one specially severe fit, and her own doctor, who administered chloroform, probably never had a more trying experience. The operation presented no difficulty, but what was specially noticeable was the marked contraction and retraction of the uterus following on the removal of the fœtus and placenta. This not only facilitated the introduction of the sutures, but markedly diminished the amount of blood loss. The child, when extracted, was dead. The uterine wall was sutured with deep and superficial silkworm gut sutures, and the abdominal wall closed by a continuous peritoneal suture of catgut, through-and-through silkworm-gut sutures not including the peritoneum, and a continuous catgut suture for the fascia. The through-and-through sutures were employed because the abdominal walls contained a considerable amount of fat. The instruments used were a scalpel, a pair of scissors, two pairs of artery forceps, a straight needle, and a curved needle. A smooth bedroom towel was cut into four pieces, which were then boiled and used as abdominal sponges. I wore rubber gloves. After the operation the fits ceased, but the patient remained in a semi-comatose condition, the pulse being very rapid and feeble. At 8 a.m. she had a fit, for which a hypodermic injection of morphia was given. This was the only fit subsequent to the operation. The chloroform did not prevent the fits during its administration. She was inclined to be restless during the remainder of the day, but passed a "fair" quantity of urine. The pulse continued rapid and she had occasional vomiting. April 22: Passed a good quantity of urine; pulse rapid; was very restless, but did not complain of pain. April 23: Took some milk and water; very restless; urine, 48 oz.; pulse very rapid; complained of indigestion; sleeping at

intervals; perspired. April 24: Good quantity of urine; not so restless; pulse not so rapid; complains of great pain in the chest; takes Benger's food well. April 25: Improving; bowels acted well; good quantity of urine, containing less albumin; pulse much slower and better; sleeps fairly well. She continued to make rapid progress. A few of the stitches in the abdominal wall were removed on April 30, and the remainder on May 2. The wound healed well. The tongue was badly bitten during the fits, producing two very deep lacerated wounds, which eventually healed under antiseptic mouth-washes, but were a cause of much pain and discomfort. The temperature on one or two occasions reached 100° F., but remained at the normal level at other times. During the convalescence she complained of indigestion and flatulence. A fish diet was given on April 30. Her nurses left on May 26. She sailed for Canada early in June, and was reported to be quite well. The urine had been normal and free from albumin since April 30.

I regret that it was not possible to record the case with greater detail, more especially the condition of the urine. Professor Osler kindly saw her the second day after the operation. She has continued to enjoy good health, but has not again become pregnant.

From a study of the literature I find that Van den Akker in 1875 is credited with being the first to perform with good result Cæsarean section in eclampsia combined with contraction of the pelvis, although one hundred years previously Lauverjat is alleged to have recommended the operation.

In 1889 Halbertsma advocated the adoption of the Cæsarean operation on the ground that it offered a good result to the mother and child, and that it influenced so much the course of the disease. He recorded three cases, all primiparæ, aged respectively 26, 27, and 23 years. The first operation was fatal, the other two were successful. He concluded that if a woman, in the last three months of pregnancy, has eclampsia, medical treatment which endangers mother and child should not be prescribed, but operation should be done at once.

Kettlitz in 1897 gave a survey of the history of Cæsarean section for eclampsia up to and including the year 1896, and found, in all, 28 cases, giving a maternal mortality of 50 per cent.

Hillman in 1899 described a case and gives the mortality in 40 cases as 52·5 per cent.

Streckeisen in 1903 made a further collection of cases and added 26 more, giving a maternal mortality in the 26 cases of 32 per cent.

Olshausen reported in 1900 that out of his last 250 cases of

eclampsia, he had performed Cæsarean section three times, two of the mothers surviving and all the children. In all three the fits ceased after the operation; the first, however, died six hours later from eclamptic coma. He advises the operation where the case is severe with a rapid succession of fits, and where labour has not commenced. When the cervix is rigid and the os closed he prefers vaginal Cæsarean section.

The *Transactions of the Edinburgh Obstetrical Society* for 1903-04, xxix, p. 194, contain records of two cases of eclampsia for which Cæsarean section was performed by Sir J. Halliday Croom. These cases appear to be the first and only examples recorded in this country.

The first was a primipara, aged 20, who was between eight and a half and nine months' pregnant. The operation, a Porro-Cæsarean section, was performed for the following reasons: (1) Her comatose condition and the rapid succession of the convulsions (they continued to recur at intervals of less than five minutes); (2) the hypertrophy of the cervix; (3) the impossibility of dilating the cervix; (4) the contracted condition of the vagina, as well as the pelvis generally. During the course of the operation there was no recurrence of the convulsions, the cyanosis was less marked, and the patient's condition generally improved. The patient died six hours later, after a severe eclamptic seizure. The cervix is stated to have been absolutely undilatable, either by fingers or instruments. Barnes's bags were quite useless, Hegar's dilators had no effect, and Bossi's instrument was not at the time known in this country.

The second case was a primipara, aged 46. Shortly before labour began she had an eclamptic seizure. The fits became worse with the onset of labour, the intervals shorter, and the coma profound. When seen by Sir Halliday Croom she had been unconscious for ten hours. The cervix was thickened and hypertrophied, projecting but slightly into the vagina, the fœtus was far above the brim, and the pelvis generally and uniformly contracted in the first degree. Dilatation was discussed and abandoned because there was no hope of dilating the cervix within reasonable time. It was important to save the child for succession reasons. Cæsarean section was performed, and a living child extracted. The mother soon regained consciousness, and remained conscious for two days, but on the third day she died from a low form of pneumonia, whether septic in origin or not remained doubtful.

These two cases with a fatal termination are the only instances recorded in this country where Cæsarean section has been tried in the treatment of puerperal eclampsia.

At the International Congress in Geneva in 1896 the opinion was expressed that "neither Cæsarean section nor forceps delivery should be regarded as ordinary operations, but are only justified when every other kind of treatment has entirely failed." If this opinion is acted upon, then Cæsarean section would always have a high mortality, for the severe cases would alone be operated on, and then only after other methods of treatment had been tried and had failed.

Let us look, however, for a moment into the question of the maternal and foetal mortality following this operation as compared with what happens after other methods of treatment. Streckeisen found in the twenty-six cases already referred to a maternal mortality of 32 per cent. Kettlitz calculated from his cases a mortality of 50 per cent. The mortality in eclampsia treated by other methods is given as about 20 to 25 per cent. The foetal mortality in eclampsia generally is between 44 and 54 per cent. Streckeisen gives in his twenty-eight cases of Cæsarean section a foetal mortality of 30 per cent., and Kettlitz a foetal mortality of 62 per cent. At present, therefore, Cæsarean section shows a high maternal mortality, and a high foetal mortality, and for this reason might be condemned. But was it not the same when this operation was first considered as an alternative to embryulcia in pelvic contraction, and when it was only adopted as a last resort when other methods of delivery had failed?

The modern Cæsarean operation has been successful, not only from improvements in operative technique, but because the indications for the operation have been more clearly defined and acted upon without delay, and before the patient has become infected through futile attempts to deliver. If this operation is to have a place in the treatment of eclampsia we must be able to say this is a case for Cæsarean section, and have the courage to act promptly, for to delay until the patient is moribund and all treatment has been a failure is to court disaster. What, then, are the indications for this operation? I would suggest the following:—

- (1) When the fits are severe and recur in rapid succession.
- (2) When labour has not commenced.
- (3) When the cervix is difficult to dilate from elongation, hypertrophy, or excessive rigidity.
- (4) When the mother is moribund, and the foetus living and viable.
- (5) When labour has commenced, and there is found considerable disproportion between the size of the child and that of the pelvis.

(6) When the surroundings of the patient are suitable for a major surgical operation, and when the services of an operator skilled in pelvic surgery can be obtained.

Eclampsia, as a rule, is not encountered before the second half of pregnancy, and becomes more frequent the nearer term is approached. Zweifel has, however, reported a case occurring in the third month. When it does occur in the latter half of pregnancy the disease is usually severe, a favourable termination generally occurring in the cases in which premature labour has rapidly supervened. In such cases, when the fits are severe and rapidly succeed one another, the indication is to empty the uterus at once, and this is best accomplished by the Cæsarean operation, the bleeding resulting from which is also beneficial. Too much time should not be spent in such cases in attempting to dilate the cervix. Unless the cervical tissues rapidly yield to the methods of dilatation adopted, it is a matter of common experience that such manipulations tend to increase the frequency of the fits and, unless the manipulations are carefully carried out, there is the further risk of septic infection. The disease being so sudden in onset, often so severe in character and distressing to behold, it may even be the means of causing the medical attendant to be less careful in his aseptic technique, as preparations may have to be made in a hurry. Septic infection is a danger which must always be prominently borne in mind, for it would appear that eclamptic patients are even more susceptible than others, and septic infection has already claimed many victims in this disease. The wearing of sterile indiarubber gloves should be made compulsory for all those who engage in the practice of obstetrics, and special precautions should be taken in eclamptic cases to disinfect all instruments used.

Whatever views may be held with regard to the first three indications for this operation, I think all will agree that when the mother is moribund, and the child alive, an attempt should be made to save the child's life, and that this is best done by a rapid Cæsarean section. Eclampsia which is associated with disproportion between the size of the child and that of the pelvis is a further indication for this operation, as in such cases it is undoubtedly the most rapid method of effecting delivery. The surroundings of the patient and the surgical ability of those in attendance are important factors in deciding what course is best to pursue in the interests of the patient. If a patient can be removed to a well-ordered hospital or nursing home, and can command the services of a competent operator, the chances of her recovery will be increased; or if her apartment is clean, and skilled assistance at hand,

the simple technique of the operation may be carried out in her own home. Should, however, the patient be in an insanitary dwelling, and no skilled surgical assistance available, her interests are best served by the adoption of expectant methods of treatment.

The Cæsarean operation under modern conditions is practically free from risk, and is in my opinion much to be preferred to the other methods of rapid delivery, including vaginal Cæsarean section. It offers, in properly selected cases, the best chance of saving the life of both mother and child, although it must be remembered that death of the foetus *in utero* is not infrequent, on account of severe convulsions.

Another important question remains for consideration, and that is—What is the effect of Cæsarean section on the eclamptic fits? The figures collected by Streckeisen show that out of twenty-eight cases, in fourteen the eclamptic fits absolutely stopped, in three the eclamptic fits were diminished, in two the eclamptic fits remained unchanged, in six no particulars, and in two Cæsarean section post mortem. Absolute cessation of the fits was observed in half the cases. It has further been observed by Dührssen and others that eclampsia ceases more frequently after artificial than after spontaneous evacuation of the uterus. Now, from what we know of the effect of labour and attempts at delivery on the frequency and intensity of the fits, we would expect that that method of rapid delivery which involves the least amount of disturbance to the uterus would be the most beneficial. Cæsarean section is undoubtedly the one which causes least disturbance, and its effect is borne out by the figures already given. But if it is only tried after other methods have failed much of its value is lost, for prolonged attempts at delivery aggravate the disease, make the patient's general condition worse, and diminish her chances of recovery.

With our present expectant methods of treatment the mortality stands at 20 per cent. to 25 per cent.; let us see what is being done to reduce it.

In Germany, Dührssen, Bumm, and others strongly favour rapid delivery, but the great difficulty in arriving at a decision as to whether active treatment should be adopted is one of prognosis. It is a difficulty which arises in the whole group of diseases associated with convulsive seizures. You cannot say for certain whether a slight fit will be succeeded by one of like severity or whether a severe fit with fatal termination may not be the sequel. If it were possible to foretell with greater accuracy the course of the disease, it would be easier to indicate what treatment should be adopted. Dührssen evidently adopts the rule,

"after the first fit empty the uterus," and this, I submit, is a perfectly logical position to assume; but we know there are slight cases which get well quickly, or if you will, readily yield to treatment, and that 75 per cent. to 80 per cent. of all cases recover. But we cannot foretell, we are prepared to take the risk, and this, I think, fairly represents our position at the present time. Bumm,¹ in a paper entitled "*Die Behandlung der Eklampsie*," gives the result of his experience of the treatment of eclampsia in Halle and Berlin. He states that he has been enabled to reduce the mortality from 25—30 per cent. to 2—3 per cent., the former high mortality existing when expectant treatment was adopted. He believes that the quicker the uterus is emptied after the onset of the fits the better the prognosis. An achievement such as this, vouchsafed for by so high an authority, is important evidence in favour of rapid delivery. As it is possible to collect statistics to prove either the advantages or the disadvantages of rapid delivery, it is, I think, more reliable if we can obtain recent evidence from a competent observer who has had a considerable experience of the disease.

Herman, who has published so many valuable papers on the subject of puerperal eclampsia, is strongly opposed to rapid delivery, and in a paper published in the *Transactions of the Medical Society of London*, 1902, xxv, p. 224, has collected a large series of statistics showing the effect of delivery on the fits. I shall, however, only refer to his own cases. During the years 1891—1901 thirty-eight pregnant women suffering from epileptiform convulsions were admitted into the London Hospital. Of the thirty-eight, twenty recovered and eighteen died. He says: "This is a very large mortality, but it is partly due to the tendency of general practitioners to send to the hospital those cases that seem likely to end unfavourably. Thus two of the patients were admitted in deep coma and died a few hours after admission. In two others there was reason to believe that the fits were due to cerebral tumours, and the cases were therefore not puerperal eclampsia in the proper sense." A post-mortem examination was unfortunately not obtained in either case.

The statistics of the Glasgow Maternity Hospital, collected by Munro Kerr² for a period of fifteen years, show a mortality of 47 per cent. Jardine says: "The majority of the patients are sent in after they have been having convulsions for many hours. It is rare for us to get them within twelve hours of the first fit." If this is so, is it not

¹ *Deutsch. med. Wochenschr.*, 1907, xxiii, pp. 1945-7.

² *Glasgow Hosp. Rep.*, 1901, iii, p. 57.

due to the current teaching in this country that expectant methods of treatment should be adopted, and that when the case is beyond hope further advice is sought, and even then a continuance of this treatment is advised? The general practitioner looks for guidance to those who make a special study of obstetrics, and amongst the latter there is still considerable disagreement over the various methods of treatment. After delivery the fits do not always cease; indeed, they may even occur for the first time during the puerperium, but they do cease in a considerable percentage of cases. It may, however, be fairly stated that the *termination of pregnancy exerts a more powerful and constant influence on the course of the disease than any method of treatment yet employed.*

Are we, then, to continue poisoning our patients with chloroform and morphia, or are we to hasten delivery? Of all forms of rapid delivery, Cæsarean section would appear to be the best in this disease for the reasons already stated, and I would invite an expression of opinion from the members of this Section on two important questions: (1) Is Cæsarean section justifiable in the treatment of puerperal eclampsia? (2) What are the special indications for this operation?

I think that in this country the time has arrived when a reconsideration of the propriety of rapid delivery (in this disease) should take place, and that definite indications should be laid down for the guidance of those who may be unfortunate enough to encounter in their practice severe cases of puerperal eclampsia.

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DISCUSSION.

Dr. HERMAN agreed with Dr. McCann as to the great difficulty of prognosis in eclampsia. One could recognize when a patient was beginning to recover, so that a favourable prognosis might be given. One could also recognize when a patient was moribund. But in the beginning of this illness, when a patient had had one or two fits and was in coma following them, it was impossible, so far as he knew, to say whether the patient would recover or not, and this being so, it was not possible to be sure that the way in which the illness ended was the result of treatment. He also agreed with Dr. McCann that Cæsarean section was the quickest and easiest way of emptying the uterus. The great difficulty of prognosis made it impossible to judge as to the effect of treatment from a small number of cases. Dr. McCann took it for granted that the first indication of treatment was to empty the uterus. He (Dr. Herman), in a paper read before the Medical Society of London in 1902 and published in the *Lancet*,¹ had collected more than 2,000 cases from the reports of different clinics, and the comparison of the cases in which the uterus was rapidly emptied with those in which it was not interfered with was to show that there was no benefit in emptying the uterus—the fits were not stopped, nor was the death-rate appreciably smaller. He had not read anything since, nor had Dr. McCann adduced any evidence in his paper, to alter this conclusion. Dr. McCann had quoted the opinions of various obstetricians to the effect that it was a good thing to empty the uterus in eclampsia, and he was glad to hear Dr. McCann's loyal appreciation of the personal merits of his fellow-workers. But opinions were not facts. When large numbers of cases of eclampsia were compared it became evident that the prognosis was not improved by emptying the uterus. The treatment of eclampsia by Cæsarean section was not new; it had been done in many cases in many hospitals before 1902 and since, and the collective mortality of cases of eclampsia treated by Cæsarean section was about 50 per cent., while he believed that Dr. McCann was correct in putting the mortality of the disease treated by expectant methods at about 20 per cent. He agreed with Dr. McCann that the mortality of an operation could be correctly estimated only when it was performed by competent surgeons, in favourable surroundings and with the best possible technique. But if it went forth as the judgment of this Section that Cæsarean section was the proper treatment of puerperal eclampsia, that would lead to its being often done by inexperienced operators under unfavourable surroundings. Even with an experienced operator, the conditions under which the operation had to be done for eclampsia were as unfavourable to perfection of technique as they could well be. It had to be done quickly, for the speed with which the uterus could be emptied was the *raison d'être* of the operation, and the operation might be interrupted by an epileptiform fit. It was certain that Cæsarean section did not stop the fits, for many cases had been

¹ *Lancet*, 1902, i, p. 1168.

recorded in which fits persisted after this operation. He did not understand Dr. McCann's remark as to the severity of the fits as a guide in prognosis. The fits of eclampsia were epileptiform in character; now and then a fit was so bad as to asphyxiate the patient and cause death, but, short of this, one epileptiform fit was very much like another. If the fits were not epileptiform he should take it that the disease was not eclampsia. He knew of no peculiarity in the fits themselves that was of any value in prognosis. He had read and heard many times of the cervix being "undilatable." To his mind that only meant that the accoucheur would not give it time to dilate. Every healthy cervix would dilate if time were allowed. He did not believe that septic or saprophytic organisms produced any effect upon the eclamptic patient different from that which they did upon other people. The eclamptic was rather more liable to septic infection only on account of the exceptional difficulty in such cases of maintaining antiseptic technique.

Dr. HERBERT SPENCER agreed with previous operators as to the difficulty of prognosis in cases of eclampsia. He did not think it was by any means proved that delivery should be at once carried out. The facts that eclampsia often was not in the least benefited by spontaneous delivery, and that it occurred for the first time after delivery, were against rapid delivery as an essential part of the treatment. His own former experience where he had delivered rapidly by dilatation of the cervix pointed in the same direction. He admitted that Bossi's good results from the use of his instrument raised some doubt on this point; but the cases could not be properly judged until details of the cases were forthcoming, as the severity of cases of eclampsia varied greatly. Amongst many doubts as to the best treatment of eclampsia he had one settled conviction, and that was that abdominal Cæsarean section was very rarely indeed called for in eclampsia, for, even if it were proved that rapid delivery was the right treatment, the patient could generally be delivered by Bossi's dilator or vaginal Cæsarean section (both of which methods, however, he regarded with some disfavour) in less time and with less danger than by abdominal Cæsarean section, which seriously endangered subsequent delivery. Bossi¹ had recently compared the results of dilatation with his instrument and abdominal Cæsarean section, and his figures showed that with Bossi's dilator the maternal mortality was only 9·45 per cent. (14 out of 148 cases) and the foetal mortality 20·97 per cent., whereas with abdominal Cæsarean section the maternal mortality was six times as great (56·9 per cent.) and the foetal mortality nearly twice as great (37·5 per cent.).

Dr. AMAND ROUTH thought that if there was truth in the view that the eclampsia of pregnancy was due to an auto-toxæmia due to altered metabolism in the pregnant uterus, the toxins entering the mother's blood through the placental circulation, it was only reasonable to believe that emptying the uterus would stop the supply of toxins. Experience and statistics alike

¹ Bossi, L. M.: "Report of Sixteenth International Congress of Medicine," Buda-Pest, 1909. Section "Obstétrique et Gynécologie," p. 259.

appeared to support this view. The cases that got well without operation were mainly those in which delivery occurs spontaneously. Zweifel gave a mortality of 28·5 per cent. in cases under expectant and 11·5 per cent. in those under active treatment. If the patient was in labour, relief could often be afforded by rupturing the membranes to relieve pressure and delivering by forceps when the cervix is dilated. If in any given case rapid delivery was indicated and the cervix was undilated and rigid he thought abdominal Cæsarean section, by which the child and placenta could be delivered in three minutes, was preferable to vaginal Cæsarean section or Bossi's mechanical dilatation of the cervix. He disapproved of any such routine treatment of eclampsia as operating "after the first fit," as Bumm proposed, but he thought there was reason to believe that Cæsarean section would be occasionally useful. He thought Dr. McCann's indications were excellent, provided the cervix was rigid and undilated, and he thought any serious diminution in urea was another indication of value.

Mr. W. D. SPANTON remarked that formerly when he came across a case of puerperal eclampsia the routine practice was to bleed on the occurrence of the first fit, then administer chloroform, and set to work to empty the uterus. The result was usually satisfactory, as the bleeding tended to relax the cervix so that delivery could soon be accomplished—usually by turning. Some of the worst cases he had seen had been *post-partum* ones; therefore he thought the adoption of Cæsarean section, as a recognized procedure in ordinary cases where other methods such as those he had referred to, or treatment by veratrum, as advocated by Professor Mangiagalli (and said to have been very successful in his hands), had not been first tried, was hardly justifiable. No doubt in those instances where the cervix was hard and undilatable, or where pelvic contraction existed, abdominal Cæsarean section would be the safest course to adopt, but it should be reserved for such exceptional cases.

Dr. W. J. GOW said that he considered delivery by Cæsarean section was justifiable in certain cases of eclampsia. He thought that with the natural termination of labour, or as the result of artificial extraction, if no great injury had been inflicted, improvement in the symptoms was frequently observed. In severe cases, patients died even after delivery, but after the uterus was emptied the chance of recovery was greater. In one case in which he had performed Cæsarean section on account of pelvic contraction, the patient was suffering from severe toxæmia, as evidenced by the presence of a large amount of albumin in the urine and the occurrence of several eclamptic convulsions. The recovery after the operation seemed in no way interfered with by the toxæmic state, so that the operation performed under such conditions does not seem to be associated with any increased risk. The high mortality of the operation undertaken in cases of eclampsia had been alluded to; but although it was true that many such patients died, it would seem that they died of the eclampsia, and not from the operation. If the cervix was not taken up, and was rigid and undilatable, delivery by Cæsarean section seemed to him the simplest and safest method to employ.

Of course such a plan of treatment was only indicated in very severe cases of eclampsia. Dr. Gow thought that vaginal Cæsarean section was quite unsuitable unless the cervix was already taken up. To empty the uterus of a patient suffering from severe eclampsia did not necessarily cure her, but he thought it distinctly increased her chance of recovery if it could be done without inflicting serious injury upon her.

The PRESIDENT (Dr. Macnaughton-Jones) said that he had, some years since, published a case in which eclampsia occurred at the fourth month. Here he was dealing with a degree of stenosis of the cervical canal, and there was great difficulty in completely dilating the uterus. The patient was under chloroform for several hours, and death occurred before he could satisfactorily empty the uterus. Under the old expectant treatment a certain number of patients died, whether that followed was bleeding, opium, or chloroform. Some of the worst cases of eclampsia he had seen occurred *post partum*. He had published in the *Lancet*, eight years since, an interesting case, the first in which he believed Bossi's dilator was used in this country. The uterus was myomatous, and the urine was thick with albumin. He succeeded in delivering with forceps, but the patient died shortly after delivery. In two other severe *post-partum* cases he had used injections of a few minims of a 2-per-cent. solution of muriate of pilocarpine, and both recovered. He believed that there was a good deal in the old clinical differentiation of the type of convulsion, as denoting its severity: hysterical, epileptiform, or apoplectiform in type, or that other clinical difference equally distinctive in prognosis—*anæmic*, *hyperæmic*, and *toxæmic*. An *anæmic* and albuminuric primipara with high blood pressure certainly presented a most dangerous form of eclampsia. Taking the whole condition of the woman into consideration, her lowered vitality, and the condition and character of her blood and lymph vessels, he agreed with Dr. McCann that she was more liable to risks of septic invasion and sepsis generally than one passing through a natural labour. As to the question of Cæsarean section, he felt, with Dr. Gow, that there were circumstances and complications under which Cæsarean section might be the only course open. We were left to the choice of abandoning the woman to her fate or giving her a chance by operation.

Dr. McCANN, in reply, said that, while thanking the members of the Section for the reception given to his paper, he expected that there would be considerable difference of opinion regarding the advisability of his proposals. He had already dwelt on the difficulty of prognosis, but there was also the effect of the geographical distribution of the disease. In Italy they appeared to have good results from the use of *veratrum viride*; in St. Petersburg Stroganoff had equally good results with morphia; whilst in Berlin their cases were so severe they had to operate. He had recently visited Berlin and had seen the work at the Charité under Professor Bumm, and for this reason had quoted his results, as it was preferable to obtain recent evidence from a reliable observer, who had sufficient material at his command, rather than quote statistics of doubtful value. Bumm had reduced his mortality of 25 to 30

per cent. to 2 to 3 per cent. through the routine adoption of rapid delivery. The mortality in this country from expectant methods of treatment still stood at 20 to 25 per cent. This, it must be admitted, was unsatisfactory, and surely some attempt should be made to reduce it. The cases of eclampsia occurring in the later months of pregnancy which recovered were generally those in which premature delivery rapidly supervened. This, then, was the manner in which nature cured the disease, and when we were in doubt we could not do better than follow her teaching. Further, if there was anything in the present theory of the placental origin of the toxæmia, the prompt removal of the fœtus and placenta was clearly indicated. Dr. Herman said, in effect, that the difficulty in dilating the cervix was in inverse ratio to the patience of the doctor. This reference served to emphasize what had been insisted on in the present communication, for it was the repeated and long-continued efforts to dilate which were so harmful in increasing the severity of the fits and the liability to septic poisoning. A special feature in the operation was the marked contraction of the uterus after delivery of the child, but, from what he had heard from those who had performed Cæsarean section for eclampsia in Berlin, this did not always occur, and there might be considerable trouble owing to hæmorrhage. As had been mentioned, Bumm's results were better than those of Bossi, referred to by Dr. Spencer. The instrument designated Bossi's dilator was not, in his opinion, a surgical instrument. He had never used it, and had no intention of doing so. It should be quietly consigned to oblivion. Dr. Spencer's reference to vaginal Cæsarean section made him wonder how often he had performed the operation. When the cervix was long and rigid, vaginal Cæsarean section became a very difficult operation, for even after the incisions had been made the child had still to be delivered. There appeared to be a good deal of misconception as to what was really meant by vaginal Cæsarean section. This operation, as performed by Dührssen, consisted in division of the anterior wall of the cervix and lower uterine segment, the posterior cervical wall, and, if necessary, the perinæum and posterior vaginal wall; whereas Bumm favoured rapid delivery by means of median hysterotomy, the anterior cervical wall and the lower uterine segment being incised. There were two important danger signals in the course of a case of eclampsia—viz., rapidity of the pulse and elevation of temperature. It was the progressive increase in the rapidity of the pulse that prompted him to evacuate the uterus rapidly. Bleeding, as Mr. Spanton had said, was the old treatment for eclampsia, and it was still one of the best methods to employ, and doubtless had an influence during the course of an operation. He fully concurred with what Dr. Gow had said regarding vaginal Cæsarean section, and was pleased to hear he was in agreement with him as to the advisability of performing Cæsarean section in certain cases of eclampsia. The case narrated was a very severe one, and there could be no doubt that the operation saved the patient's life. He confessed to being an enthusiast regarding the use of sterile india-rubber gloves, and considered they had formed an important advance in aseptic technique.

A Contribution to the Life-history of Fibromyomata of the Uterus.

By ARTHUR J. WALLACE, M.D.

(ABSTRACT.)

THE paper relates the histories of ten cases of "fibroids" of the uterus, observed over periods of years varying from three and a quarter to twenty-eight. In Cases II to X the observations were made partly by my father, the late Professor John Wallace, partly by myself, the dividing line between the two sets being the year 1898. It should be stated that my father was most strongly opposed to operation in cases of fibromyomata unless danger to life were threatened. After his death nine patients confided to me the continuance of the palliative treatment he had initiated.

Of the ten cases, complete disappearance of the tumour took place in two.

CASES IN WHICH COMPLETE DISAPPEARANCE OF THE TUMOUR OCCURRED.

Case I is that of a married lady who was first brought to see me by Dr. A. C. Wilson, of Formby, on July 6, 1904. She was at that time aged 42, was well nourished and had an excellent colour. The youngest of her four children was aged 11 years. For twenty years she had suffered from prolapsus uteri, for which a ring pessary had been worn continuously, except during her pregnancies. For some time she had been a sufferer from mitral regurgitation and dilated heart, and was at times completely prostrated by attacks of cardiac weakness, indeed on several occasions Dr. Wilson had feared that death was imminent. He, having had the patient under close observation for nine years, had of late noticed that a gradual rise in the pulse tension was taking place, and, as the result of observation, thought he had formed the hypothesis that there existed a relation between increased arterial tension and the development of neoplasms. For this reason he kept a very watchful eye upon Mrs. V., and, as the ring pessary required attention every few months, his observations included the state of the pelvic organs. In January, 1904, an enlargement of the uterus was detected. Menstruation had always been profuse, the periods lasting seven days, and recurring every twenty-one days, but in April, 1904, the losses became excessive. In June Dr. Wilson made another pelvic examination, and was astounded at the increase in size of the uterus. In July, when I saw the patient, she complained of enlargement of the abdomen, menorrhagia, profuse leucorrhœa, and severe headaches. On examination a hard, smooth-surfaced tumour occupied the lower half of the abdomen:

its upper limit reached the level of the umbilicus, and the greater part of its bulk lay on the right side. Transversely it measured 6 in., while the distance from the pubic crests to the upper border of the tumour was $6\frac{1}{2}$ in. Two smaller nodules were felt at the lower part of the mass, and the inferior of these could be felt *per vaginam* through the anterior vaginal wall. The supravaginal cervix ran into the mass and became incorporated with it, movement of one being communicated to the other. Bimanually the mass could be moved about to a limited degree. The uterine appendages could not be recognized. The kidneys and other vital organs, with the exception of the heart, were found to be healthy. A diagnosis of multiple fibromyomata of the uterus was made, Dr. Wilson concurring. Operation was advised against at that time, in view of the serious cardiac condition, and treatment was directed to the latter and to controlling the menorrhagia. On June 14, 1905, I saw the patient again. She had undergone Nauheim treatment in London, and, so far as the heart was concerned, considerable improvement had resulted. The tumour, however, had increased steadily in size. Latterly pain had been experienced over an area on its right side, and some tenderness on pressure was elicited over the same situation. The transverse measurement of the tumour mass was now 8 in., and its upper border lay 7 in. above the pubic crests. Its outline was much more rounded, and the lower abdomen was practically filled by it. Menstruation had continued regularly, as profusely as ever, but each period now lasted five days in place of seven. Drugs had been used without in any way lessening the menstrual losses. I did not see the patient again until the present year, but Dr. Wilson informs me that in 1905, under treatment, the blood pressure became less, and then he noticed that the tumour was gradually diminishing in size. In 1907 its upper border was "2 in. below the umbilicus," and about this time menstruation began to be irregular, occurring at intervals of two to three months, the losses being still very profuse. The patient developed also glycosuria to the extent of 8 to 10 per cent. Under a diabetic diet recovery from this took place in six months. In 1905 the pulse tension had begun to lessen and the headaches to disappear. In the winter of 1908-9 the patient suffered from feverish attacks, each of which lasted about ten days, the temperature rising in the evenings to 101° F. or 102° F., and falling in the mornings to 100° F. Dr. Wilson noted that after each feverish attack the tumour became sensibly smaller. There was a vaginal discharge at this time, but it was never great in amount. The patient was wearing a ring pessary, and the discharge was similar in appearance and odour to the leucorrhœa found in those who wear rubber pessaries. It never came away in any quantity, and was only sufficient to keep the vulva slightly moist. In June of last year (1909) the patient was in London, and, being at that time greatly troubled by hæmorrhage, she consulted Dr. Herbert R. Spencer. Through his courtesy I am able to mention that he found the patient possessed at that time "a multinodular fibroid uterus as big as the pregnant organ at the fourth month." In September of last year (1909) the patient came to see me after an interval of over four years. On examination I found the uterus slightly

larger than the size of the normal parous organ. Its surface was smooth and without inequalities or irregularities to indicate the previous existence of fibroids of considerable size. The uterus itself was freely movable, indeed it tended to prolapse without the support of the ring pessary, which was still being worn. The disappearance of the fibroids was so astonishing and so perplexing that I spent a considerable time in palpating the uterus, but each investigation only confirmed the first, that the fibroids had quite vanished. It may be added that Dr. Wilson has since confirmed my observation. The patient's general condition had greatly improved; the headaches had gone, the cardiac trouble was very much better, and the pulse tension had lessened considerably. Menstruation still occurred at intervals of from two to three months, and the losses were described as being very profuse and accompanied with clots.

Case II is that of a single lady who, when aged 36, was sent in August, 1889, by Dr. Samuel Cookson, who at that time practised in Stafford. At this lady's first visit the pelvic cavity was occupied by a fibroid which sprang from the posterior wall of the uterus, and its pressure had led to constipation and dysuria ("stoppage of bowels and bladder"). The cervix was displaced forwards and lay immediately behind the symphysis. The uterine sound could be passed easily through the patulous os in a direction vertically upwards for a distance of $3\frac{1}{2}$ in. The menstrual periods lasted from four to six days, and recurred at intervals of from twenty-one days to twenty-eight days, more often twenty-one. The tumour and uterus were there and then pushed up into the abdomen, but, unfortunately, no note was made as to how high above the pubic crests the upper border of the mass then lay. The patient was advised not to submit to operation, a view which was endorsed by Dr. Cookson. In December of the same year—i.e., four months later—the tumour mass reached "three finger-breadths above the pubes"; it was "much smaller," but was "not in the pelvis." Early in 1890 the tumour still reached a height of "three-finger-breadths" above the pubes, but by August of the same year it had become small enough to drop back into the pelvic cavity, so that it could no longer be felt abdominally. Its size was noted to be that of "a medium orange," and it was movable with the uterus. Its base of attachment had become less extensive, and a sulcus now separated its superior surface from the upper part of the posterior uterine wall. In 1891 it was noted that diminution in size had continued in slight degree. Retroversion of the uterus now occurred and caused dragging pains. These were relieved after a ring pessary had been placed. During the years 1892-97 the patient presented herself at regular intervals in order to have her pessary renewed, and therefore ample opportunities for watching the fibroid occurred. In this period of seven years there occurred a slight decrease in size, from "a small orange or less" to a "small mandarin orange." In 1899 the tumour was rounded and it had a diameter which I estimated as being about $2\frac{1}{2}$ in. There are fallacies connected with the estimation of the size of a pelvic swelling, either in inches or by comparison, nevertheless a diameter of $2\frac{1}{2}$ in. or comparison to a "small orange"

gives a fair idea of the bulk of the growth. It had a sessile attachment to the posterior wall of the uterus by a broad base, and both tumour and uterus could be moved as one mass. The uterine appendages were normal in all respects. The menstrual periods occurred too frequently and lasted too long, a condition which persisted until 1902, when indications of the approaching menopause appeared, the patient being at this time aged 49. The final menstrual loss occurred in the following year, 1903. From 1899 onwards very little alteration in the size of the growth was noted: if any change took place, it was a diminution that could be appreciated only bi- or triennially. In 1908 the growth was quite sessile on the uterus and its diameter was, roughly, $1\frac{1}{2}$ in. The uterine cavity measured $2\frac{1}{2}$ in. by the sound. Early in 1909 a projection from the posterior wall of the uterus represented the tumour, and in July only a slight irregularity could be recognized at what was formerly the site of a growth of considerable size. When examined early in the present year (1910) not even the slightest irregularity could be detected, although it is possible that, if the site were to be cut down on, some fibrous thickening, or even a small fibrous nodule, might be found. Still, from the clinical point of view, the fibroid has ceased to exist after a life of at least twenty years.

CASES IN WHICH SHRINKING ONLY OCCURRED.

Case III.—Mrs. D., first seen in 1890, when she was aged 54. The lower half of the abdomen was occupied by a hard, bilobed tumour. In 1891 the tumour had diminished "to a half of its former size." In 1893 a lump about the size of an English walnut could be felt on the left side of the lower abdomen; it was attached to the uterus by a pedicle. The uterus was $3\frac{1}{2}$ in. long by the sound, and had the contour of the normal organ. This patient was alive and well in 1908, and had had no further pelvic trouble. With the exception of two "floodings," menstruation had always been normal; it ceased at the age of 54.

Case IV.—Miss W., aged 44. In 1891 the greater part of the abdomen was occupied by a large, hard tumour, with an irregular surface. On the right side its upper border was distant "four fingers' breadth from the ribs," on the left a projection passed into the loin. The tumour was mobile; *per vaginam* the cervix could not be located, but on the surface of a hard mass, lying in Douglas' pouch and bulging forward the posterior vaginal wall, there was found a slit, through which the sound passed $3\frac{1}{4}$ in. Three months later the upper border of the tumour lay at the level of the navel, and six months later $1\frac{1}{2}$ in. below it. In 1898 the tumour could not be recognized abdominally; it lay in the pelvis, projecting from the posterior uterine wall, and had the size of "a mandarin orange." Eight years later it had become calcified. Prior to the menopause, at the age of 45, menorrhagia had existed.

Case V.—Miss T., aged 51. In 1892 the lower abdomen was occupied by two swellings that were connected together, that on the left being a fibroid, the upper extremity of which reached the level of the umbilicus. The sound passed

3½ in. Steady diminution in size occurred up to 1900, when the tumour had the size of the head of a seven months' foetus. Abdominal pain and tenderness was more or less continuously present for fourteen years, thirteen of which were post-climacteric years. In 1909 the uterus and tumour together formed a swelling the size of a hen's egg.

Case VI.—Mrs. D., aged 43, had in 1892 an irregular abdominal tumour, reaching up to midway between pubes and umbilicus; *per vaginam* the left lateral fornix was obliterated by a mass bulging down from above. The sound passed 4½ in. In 1894 the upper border of the tumour lay 2 in. above the pubes, in 1899 1½ in. above, and in 1900 the fibroid lay entirely in the pelvis. It had then the size of a Jaffa orange, and remained unchanged until the patient died, in 1906, from carcinoma of the rectum. The menopause had occurred in 1898, at the age of 50.

Case VII.—Mrs. F., aged 33, had in 1892 a hard, ovoid abdominal tumour, reaching up to a point midway between pubes and umbilicus. The sound passed 5 in. The tumour decreased in size until 1895, when its upper border lay 1½ in. above the pubes. From 1897 it steadily increased in size until 1904, by which time the enormous tumour occupied almost the whole abdomen. General symptoms were absent, and Dr. C. J. Cullingworth agreed that operation could not be pressed. Shrinking began in 1904, and continued steadily. In 1907 the menopause occurred at the age of 48. At the present time (1910) the upper border of the growth lies 1 in. above the navel, and the mass occupies about half of the width of the lower abdomen.

Case VIII.—Mrs. N., aged 49, in 1895, had at that time a mass of multiple fibroids reaching up as far as the umbilicus. In 1900 the upper border was halfway between pubes and umbilicus, and since that time no further change occurred up to 1905. In 1909 the patient stated by letter that the tumour had ceased to give trouble "since some time past." The menopause occurred in 1897, at the age of 51.

Case IX.—Miss R., aged 25 in 1872, when treatment for retroversion was carried out by the late Dr. Thorburn, of Manchester. In 1882 (age 35) he recognised a fibroid in the posterior uterine wall. The tumour grew until its upper border lay, in 1893, halfway between pubes and umbilicus, and still higher in 1897. It remained in this condition until 1902, since when there had been no opportunity of further examination. The patient wrote in 1910 that she "could feel a lump about the size of a lemon, and of oblong form, rather to the left side of the abdomen." The menopause occurred in 1899, at the age of 52.

Case X.—Miss F., aged 31, in 1886. She was at that time under treatment for retroversion, and seven years later developed fibroids. These grew large enough to fill the pelvis in 1899, but had begun to decrease in 1901. In 1910 the uterus was senile, with a small nodule on its anterior, and a sessile, subserous fibroid attached to the posterior wall.

ANALYSIS.

Parity.—Five patients were single and nulliparous (Cases II, IV, V, IX, and X). One (VI) was a barren widow, one (VII) had had an abortion prior to the appearance of her tumour, one (III) had had one child, and two (I and VIII) had had several children.

Alterations in Menstruation.—Menorrhagia occurred in Cases I, II, IV, VI, VII, and IX :—

Case			Occurrence of menorrhagia
I	During whole period of five years menstruation still profuse, although irregular
II	Menorrhagia for three years prior to menopause
IV	For twelve months preceding menopause at age of 45
VI	Menorrhagia and metrorrhagia up to eighteen months before menopause at age of 50
VIII	Menorrhagia at a time two years before menopause at age of 51 (data incomplete)
IX	Menorrhagia for three years, then profuse menstruation for nine years till menopause occurred at age of 52

In one case (VIII) the menstrual periods were scanty and lasted two days when the patient first came under observation. At this time the fibroid reached half-way to the umbilicus. Later, when the health had improved, the periods lasted seven days. In the other cases (III, V, and X) menstrual alterations were absent or trifling.

Periods during which the Cases were under Observation.

Case			Period of time
I	Five years
II	Twenty years
III	Three and a quarter years; reported well sixteen years later
IV	Nineteen years
V	Seventeen years
VI	Thirteen years
VII	Eighteen years; still under observation
VIII	Ten to fifteen years
IX	Twenty-eight years
X	Twenty-four years

Growth of the Tumour to the Maximum was observed in four cases : Case I, in which growth was so rapid as to warrant the term "acute"; and Cases VII, IX, and X, in which the increase was slow—chronic. In three cases the fibroids were watched from their earliest beginnings, the opportunities occurring because all three patients were under treatment for uterine displacements prior to the appearance of the myomata. Case I suffered from prolapsus, Cases IX and X had troublesome retroversions. In the other six cases the growths had attained their maximum size before or when they were first examined.

Shrinking of the Tumour.—Incomplete shrinking occurred in eight out of the ten cases. A noteworthy point is the rapidity with which it took place at first in Cases II, III, and IV. In Case II there occurred in the course of eighteen months a decrease to almost one-fifth of the original size, so that the tumour becomes once more a habitant of the pelvis. Yet eighteen and a half more years had to pass ere the growth finally disappeared. In Case III, during a period of eleven months after the first examination, a decrease to less than half the original size took place; and in Case IV, during the ten months following the first examination, a decrease to nearly half of the original size. Whether these decreases depended on genuine absorption of the component elements of the tumour is questionable; some such absorption may have been in progress, but there is always the possibility of lessening in bulk by reason of reduction of œdema. This is supported by the circumstance that in Cases II and IV chloride of calcium was being taken, and this salt is reputed to possess the power of abating œdema. I believe œdema explains certain temporary alterations in size that were observed in Cases V and VI. In Case VI it was repeatedly found that within short periods of time the size of the tumour varied, and similar changes were also noted from time to time in Case V.

Relation of Shrinking to the Menopause.—In Case I disappearance occurred before the menopause had taken place, but the latter must undoubtedly have commenced to exercise an influence, since menstruation, although profuse, had become irregular.

Case	Menopause occurred at	Shrinking of tumour began	Result
I	—	Before menopause, and was complete before menopause	
II	49	Twelve years before menopause ...	Completed to zero after menopause
III	56	About the time of the menopause ...	Incomplete
IV	45	A few months before the menopause ...	„
V	52	Shortly after the time of the menopause	„
VI	50	Six years before the menopause ...	„
VII	48	About the time of the menopause ...	„
VIII	51	„ „ „ „ „	„
IX	52	„ „ „ „ „	„
X	46	One year after the menopause ...	„

In closing the discussion on his paper read before the Obstetrical Society in 1893, Alban Doran mentioned that Skene Keith seemed correct in stating that fibroids did not begin to diminish until two years after the menopause. Of the nine “chronic” cases I report, in

three the tumour began to shrink before the menopause, in three the shrinking coincided with the onset of the menopause, and in the remaining three the diminution began after its onset. Of the latter three (Cases III, V, and X) in no instance was the commencement of shrinking delayed for more than one year after the menopause.

Cardiac Disease.—Valvular disease existed in Cases I, VII, and VIII; dilatation and hypertrophy of the left heart in Case IV. The points in the several cases are as follows:—

CASES OF VALVULAR DISEASE.

Case I.—(a) The patient suffered severely from mitral regurgitation prior to detection of the fibroid. (b) With its development, and the onset of menorrhagia, the heart trouble became at first exaggerated, but subsequently improved under special treatment, although the fibroid continued to grow. (c) Notwithstanding the severe menstrual losses anæmia did not supervene.

Case VII.—(a) When the patient was first seen there were present both a fibroid and mitral regurgitation. (b) The patient was markedly rheumatic. (c) During the growth of the tumour to its maximum, the cardiac condition did not cause any symptoms. (d) Mild symptoms of a temporary kind (slight shortness of breath on exertion) occurred several years after shrinking of the tumour had begun.

Case VIII.—(a) When the patient was first seen, she had a large fibroid uterus, but no cardiac disease existed, although menorrhagia had led to deterioration of the general health. (b) Pre-systolic and systolic murmurs developed after the menopause.

CASE OF DILATATION AND HYPERTROPHY OF THE LEFT HEART.

Case IV.—(a) The tumour continued to shrink from the time the patient was first seen. (b) During the early and middle periods of observation no cardiac disease was detected. It developed after the tumour had dwindled to a size that permitted it to become a pelvic habitant, and after calcification had occurred.

Of the ten cases, therefore, it is doubtful whether even in one of them the tumour can be charged with any causative influence in the production of heart disease. Nine of the ten were instances of women who for years had struggled along with their tumours, and one would expect in such cases that cardiac disease would certainly develop if there were any real foundation for the view that fibroids tend to produce cardiac lesions. Thomas Wilson found that out of seventy-two cases of fibroids submitted to operation, “there were objective signs of more or less severe affections of the heart in thirty-three, or nearly 46 per cent.

TABLE OF

Case	Married or single, nullipara or parous	Age when fibroid was first noted, and date	Menstrual data	Previous gynecological history	Data regarding fibromyomata	Growth of tumour to maximum observed for
I	Married, four children	42, Jan., 1904	$\frac{7}{21}$ profuse, later excessive	Prolapsus uteri for 20 years	Multiple; interstitial and subserous; mass occupied lower half of abdomen, and the portion palpable measured 6 in. by $6\frac{1}{2}$ in.; later, 8 in. by 7 in.	1 year
II	Single, nullipara	36, Aug., 1889	$\frac{4-6}{21-28}$ usually, "a little too often"; no floodings	Nil	Interstitial at first, then sessile subserous; incarceration of tumour in pelvis, constipation and dysuria; three months after release the upper border of the tumour reached "three fingers' breadths above pubes"; sound passed $3\frac{1}{2}$ in.	Maximum attained when first seen
III	Married, one child	54, May, 1890	$\frac{6}{21}$ regular till Sept., 1889	—	"Lower half of abdomen occupied by a hard bilobed tumour, the right half of which was larger than the left"; the variety of tumour in the first instance = doubtful, the last form = pediculated, subserous	Maximum had presumably been attained when the patient came under observation
IV	Single, nullipara	44, July, 1891	$\frac{4-5}{28}$ regular	—	Greater part of abdomen occupied by a large, hard, irregular tumour, which projected deeply into the left loin, and also sent a projection down into the pelvis; the first form = ?; the final = pediculated subserous; the sound passed $3\frac{1}{2}$ in.	Ditto
V	Single, nullipara	51, Feb., 1892	Irregular menstruation; only five periods in 1891	Right iliac pain	Lower abdomen occupied by two swellings which were connected together, that on the right being the uterus, the left a fibroid; sound passed $3\frac{1}{2}$ in.	Ditto
VI	Widow, no children	43, Aug., 1892	$\frac{7}{28}$ regular, profuse	Menorrhagia, abdominal pain and swelling	An irregularly-shaped tumour reached half-way to umbilicus, and from it a projection downwards obliterated the left fornix; sound passed $4\frac{1}{2}$ in.	Ditto
VII	Married, widow since, one abortion in 1887	33, Aug., 1892	$\frac{2}{28}$ scanty; later $\frac{7}{28}$ regular	Never well since abortion in 1887	A hard ovoid tumour reached from the pubes to a point midway between pubes and umbilicus, and projected downwards into pelvis; sound passed 5 in.	Tumour lessened in size till 1895, increase found in 1897, maximum attained in 1904; 12 years
VIII	Married, widow since, several children	49, 1895	—	Menorrhagia and abdominal swelling	Lower abdomen occupied by a mass of multiple fibroids reaching almost to umbilicus; no pelvic projection; variety probably interstitial and subserous	Maximum attained when first seen

CASES.

History of tumour after its maximum size had been attained	Relation of first shrinking of tumour to menopause	General conditions present during "fibroid life"	Special conditions connected with fibromyomata	Case under gynæcological observation for	Meno-pause attained at	Subsequent health
Total disappearance; slowly at first, finally from size of 4 months' pregnant uterus to zero in 4 months	Before the menopause	Mitral regurgitation and dilated heart, high systemic blood-pressure, glycosuria, enlargement of thyroid	Menorrhagia, febrile attacks, each followed by diminution in size of tumour	5 years	Probably occurring now (1910)	Good; prolapsus uteri
Slow, gradual disappearance, extending over 20 years; pre- and post-climacteric	12 years before the menopause	None	Retroflexion	20 years	49 (1902)	Excellent; retroversion persists; wears pessary
Eleven months later tumour had diminished to one-half its former size; in August, 1893, only a pediculated lump, the size of an English walnut, remained	After the menopause	None	"Floodings" twice in 1890	3½ years; in 1908 the patient's doctor reported good health	57 (1893)	Good
Steady shrinking to minimum size ("a mandarin orange") in 1898; calcification occurred later	A few months before the menopause	Hypertrophy and dilatation of heart; no valvular disease; dyspnoea on exertion	Menorrhagia for 12 months	19 years off and on	45 (1892)	Good, except for the cardiac trouble
Shrinking, with temporary variations in size; in 1909, uterus and tumour together = hen's egg size	Shortly after the menopause	Nervous irritability, the result of more or less continuous pain; hæmorrhoids	Abdominal pain and tenderness	17 years	52 (1893)	Poor; troubled by pain for 13 years after the menopause
Shrinking, with slight temporary variations, to the size of a Jaffa orange	6 years before the menopause	Dyspepsia, hæmorrhoids, obesity, poor health, shingles; death from carcinoma of rectum in 1906	Menorrhagia, metrorrhagia, retroflexion	13 years	50 (1898)	Poor; semi-invalid
Steady shrinking (<i>vide</i> diagrams)	About the time of the first indications of the onset of the menopause	Mitral systolic bruit, high systemic blood-pressure, rheumatism, enlarged thyroid	Discomfort, caused by size and weight of tumour	18 years	48 (1907)	Good
Shrinking until 1905, when the upper border of the mass reached half-way to the umbilicus; part of mass in pelvis	Probably with the menopause (doubtful)	Mitral stenosis and regurgitation	Pressure symptoms (bladder), menorrhagia	10 (to 15) years	51 (1897)	Fair; has now (1910) double cataract, and is almost blind

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TABLE OF

Case	Married or single, nullipara or parous	Age when fibroid was first noted, and date	Menstrual data	Previous gynecological history	Data regarding fibromyomata	Growth of tumour to maximum observed for
IX	Single, nullipara	35, 1882	3 28 later 7 28 "floodings"	Retroversion of uterus, 1872-1885	The tumour was detected whilst the patient was under treatment for retroversion, and was watched until its upper border reached a point corresponding to the junction of the upper and middle thirds of the measurement between pubes and umbilicus, the lower abdomen being filled up by the mass	Growth observed from zero, in 1888, to the maximum in 1897; 9 years
X	Single, nullipara	43, 1898	1-2 28 "scanty"	Retroversion of uterus, 1886-1910	Two small fibroids were discovered in 1898; next year (1899) the pelvic cavity was filled by the enlarged uterus; the final form of the main tumour = subserous	Observed from zero to maximum (1898-1899); 1 year

Of these, however, twelve had murmurs, probably hæmic." Six had valvular disease, fourteen myocardial affections, and one adherent pericardium. Wilson maintains that these lesions depended largely on the fibroids, and similar views have been advanced by other writers. On the other hand, Howard Kelly and Cullen¹ state: "Some authorities

Condition of each Patient prior to and after the Menopause.

Case	Health prior to menopause	Health subsequent to menopause
I	Semi-invalidism to invalidism; prolapsus uteri; cardiac	Good; prolapsus uteri
II	Fairly good	Excellent
III	Fairly good	Good
IV	Good enough to permit patient to follow teaching profession	Good
V	Poor; abdominal pain and tenderness from time to time; nervous irritability	Abdominal pain for thirteen years after menopause
VI	Indifferent, with occasional periods of fair health; menorrhagia	Indifferent up to death from carcinoma recti
VII	Good, despite rheumatism and cardiac	Good
VIII	Poor; cardiac	Fair; tumour gives no trouble; double cataract and blindness
IX	Indifferent; abdominal pain and tenderness; menorrhagia; gout	Invalid
X	Poor; retroversion; pelvic discomfort	Semi-invalid; neurasthenia; retroflexion

¹ Kelly and Cullen, "Myomata of the Uterus," Philad. and Lond, 1909, pp. 452, 453.

CASES (continued).

History of tumour after its maximum size had been attained	Relation of first shrinking of tumour to menopause	General conditions present during "fibroid life"	Special conditions connected with fibromyomata	Case under gynecological observation for	Meno-pause attained at	Subsequent health
Slow, gradual shrinking; a small part of the remainder can still be felt on abdominal palpation	Probably with the menopause (doubtful)	Gouty manifestations, neuroses, circulatory disturbances (functional)	Abdominal pain and tenderness, menorrhagia	28 years off and on	52 (1899)	Invalid
Slow, gradual shrinking; the larger fibroid had finally the size of a bantam hen's egg	A decrease was noted 1 year after the menopause	Neurasthenia, "nervous breakdowns," sciatica	Retroversion; pelvic discomfort due (?) to weight of uterus and tumour	24 years off and on	46 (1899)	Semi-invalid; retroversion persists; wears pessary

claim that the myoma in itself brings about cardiac changes. If such were the case, then the larger the myoma the more pronounced would be the cardiac murmur. This has not been our experience. The largest tumours have not been associated with any cardiac symptoms, but the heart complications have almost invariably been associated with copious bleeding from the uterus. Most of the murmurs noted in our cases were, at the time, considered to be functional."

Of ten patients (*see* Table, p. 218) two had fairly good health up to the time of the menopause, and two moderately good. The other six had either poor or indifferent health. After the menopause the health was good in four, but of these one had lost her tumour. Another (Case I), who had also lost her tumour, enjoys good health, but she has not yet attained the menopause. Of the remaining five, one died after a long period of ill-health and suffering; one is still troubled by abdominal pain; one leads a life of invalidism, another of semi-invalidism, whilst the last obtains such enjoyment from life as a sightless old age can offer.

To my thinking this recital constitutes a serious indictment of the application of palliative treatment to cases of uterine fibromyomata. Even in our own profession still lingers the legend that after the menopause fibroids will disappear, or at least cease to trouble. Pathology had done much to expose its fallacy. From the clinical side nine of the cases related illustrate the trials and discomforts endured by the lifelong possessors of these tumours. Viewed from this standpoint alone,

palliative treatment appears inapplicable to cases in which fibroids cause symptoms and the condition of the patient's general health justifies operation.

DISCUSSION.

Dr. HERBERT SPENCER expressed his surprise that the tumour in Case 1, which, when he examined it, was as big as the pregnant uterus at the fourth month, should have completely disappeared in a few months. Degenerated fibroids might become cystic, and might rupture into the peritoneum or into the vagina, and fibroid uteri might be complicated by hydrometra or hæmatometra, and on the discharge of the fluid might "disappear"; but there appeared to have been nothing of the kind in Dr. Wallace's case. The paper was a valuable record of carefully observed cases, but he did not understand why the author drew the conclusion that all fibroid uteri should be operated on. He thought the cases pointed in the opposite direction.

Dr. AMAND ROUTH had seen uterine fibroids spontaneously disappear after the menopause. He mentioned a patient whom he had seen first in 1890, aged 47, who had had an enormous fibroid tumour for fourteen years, with severe menorrhagia and pressure symptoms. She refused operation, but had been to Kreuznach for the last six years. She soon became bed-ridden, and hæmorrhage became continuous, and in 1894 rigors and tenderness made it probable that degeneration was taking place. The uterus began rapidly to shrink in 1895, but the menopause did not occur till 1903, when the patient was 60 years old. Slight hæmorrhage recurred in 1906, due to a small mucous polypus; in 1909 the fibroid had disappeared. This, and cases quoted by Dr. Wallace, only proved the desirability of radical operation when serious symptoms were developing, for this patient was a hopeless invalid for over twenty years, and barely escaped with her life.

The PRESIDENT (Dr. Macnaughton-Jones) said that these cases of spontaneous disappearance of myomata were very interesting from the histological point of view. He was glad, however, to hear Dr. Wallace's opinion that the histories of these eight cases, which were spread over a period of eighteen years, were not to be taken as affording any argument against operation for myoma. They had now the details of some eight thousand clinical histories and post-mortem appearances collected through the statistics of Winter, Charles Noble, Ellice Macdonald, and Tracy. These clearly proved that the dangers after from 40 to 50 years of age increased directly in proportion to age, and it was perhaps an under-statement of the respective averages to say that carcinoma was present in 2 per cent., sarcoma in 3 per cent., necrosis in 6 to 7 per cent., and diseased adnexa in over 30 per cent. of myomatous tumours. This did not include various other complications, such as involvement of the appendix and those due to pressure or implication of the ureter and kidney.

Obstetrical and Gynæcological Section.

June 9, 1910.

Dr. H. MACNAUGHTON-JONES, President of the Section, in the Chair.

The Pelvic Conditions resulting from the Slighter Forms of Puerperal Sepsis, and their Treatment.

By Sir WILLIAM JAPP SINCLAIR, M.D.

THE subject to which I wish to call your attention is perhaps best illustrated in the out-patient departments of our gynæcological hospitals, and the most striking cases are those of "one-child sterility." The patients belong, for the most part, to the class who even yet receive the least possible attention during labour and the lying-in state, and who most patiently endure the discomforts and ailments which can ultimately be traced directly to parturition and the puerperium. The category of cases with which I wish to deal contains those which in the puerperium are not diagnosed or abnormal, or, even in the ordinary course of medical practice, are not definitely diagnosable.

The term "morbidity," which we have accepted with a certain degree of levity, and with entire confidence that we comprehend its causes and its consequences, is responsible for a large proportion of the chronic invalids who so patiently endure the travesty in gynæcological practice which characterizes so much of our out-patient work. Much harm may be done in the female pelvis with the temperature under 100·4° F., and without a quickening of the pulse sufficient to attract the attention of the midwife, or even of the busy general practitioner.

Why are these cases not diagnosed ?

(1) The ordinary midwife makes no exact observations. She is incapable of doing such a thing. The number of the *bona-fide* type of persons practising midwifery who cannot read a clinical thermometer is astonishing and deplorable, and this, seven years after the rules drawn up by the Central Midwives Board have come into force.

(2) Our "monthly nurses" belong largely to the same uneducated class. They miss the slighter symptoms, and consequently at the daily visit give the practitioner an unduly favourable report of the patient's condition. It is so much more pleasant and easy to do so.

(3) The most careful and experienced medical practitioner, with an apparently normal case, visits only once a day, and that in the forenoon. The pulse and temperature are then at their best, and if the pulse is quickened in some measure the acceleration is attributed to excitement due to the doctor's visit. It is natural for the medical attendant to assume that when everything known to obstetric science has been done for the welfare of the patient the puerperium will be normal, unless observations are made to the contrary. We know, of course, from painful experience that absolute asepsis of the genital organs is as yet impossible of attainment in private practice, especially among the working classes, but with our well-founded belief in cleanliness and non-interference, and our practice in conformity, we obtain very good results upon the whole. Fortunately, an encyclopædic knowledge of bacteriology is not so generally diffused among practitioners as to paralyse our efforts at prophylaxis and treatment, as has been the case to such a large extent in some regions of the Continent, especially in Germany.

In contrast with private practice it is alleged that "morbidity" is unknown in some of our lying-in hospitals. Their statistics should be exactly inquired into with some salutary scepticism. For the sake of peace I shall not compare British lying-in hospitals in this respect, but if, for illustration, we look at the reports of a series of German obstetric clinics we are at once struck with the extraordinary range between the maximum and minimum of morbidity. The differences appear to depend not upon facts but upon interpretation of terms, and also largely upon the temperament of the director of the hospital. In the class of case in British practice to which I am seeking to attract your attention, neither pulse nor temperature observed by the nurse may amount to "morbidity" in any sense yet generally accepted. The case is ultimately diagnosed by the remote effects of certain pathological processes.

What, then, are the symptoms which might be observed by the exercise of great care? Never a rigor. Rigor always implies sepsis of the graver degree. We can exclude all the forms and degrees of sepsis to which we attach the name of "puerperal fever," a good old term which has a prescriptive right to survive. In the severer cases still within the category which forms my subject, probably on careful inquiry it would be found that a certain sense of chilliness would be mentioned by the patient. In my experience, however, this is one of the important symptoms that is almost always glibly explained away by the nurse, and receives vastly too little attention.

We have fallen into an evil custom of manufacturing new terms not based on new knowledge, and reputations not being made on the diligent use of the Greek dictionary, as in the middle of last century. There has been a vast amount of experience and much controversy throughout the generations, but no noteworthy new knowledge has been added to obstetric science and practice since the middle of the eighteenth century, except the evangel of Semmelweis, explained by the work of Pasteur, and practically applied by its apostle Lister. Keeping this in mind, it will be seen that my subject occupies a distinct place in the new knowledge as dealing with the slighter forms of *lymphatic sepsis*. It excludes, a fortiori, the pyæmic form which is always grave even in its slighter manifestations, and the mixed hæmic and lymphatic form which shows itself more or less tardily in a comparatively light attack of *phlegmasia alba dolens*. Among the new terms bandied about without clearness of thought are "sapræmia" and "septicæmia"—responsible for many a disaster.

If we now analyse the positive signs and symptoms in the cases under consideration, we find invariably with exact and painstaking investigation (a) Accelerated pulse-rate; (b) Slight and not evanescent rise of temperature at some period within the twenty-four hours, usually in the early part of the evening—this, altogether apart from the physiological maximum and minimum, in persons who live regular and peaceful lives. For observations on these points the doctor must depend upon a well-trained conscientious nurse who is able and willing to keep full nursing notes, showing the exact temperature every four hours during the day. When the notes are a correct record the medical attendant will see at a glance during his daily visit whether there is any abnormality, and he will take measures accordingly, or resolve to make exact observations later. If the doctor belongs to the type of easy-goers, who can be satisfied with the monthly nurse's explanation

that the baby was fractious, that the husband was not very kind, or that the breasts were just "a bit too full," then so much the worse for the patient.

Is pain a symptom? There is none which may not be, and usually is, mistaken for some incidental and entirely fortuitous discomfort. The expression of pain depends so largely upon the patient's upbringing and temperament. Upon the whole, among the humbler class of patients, there is an expectation of great tribulation in the first labour and child-bed, and the fear of being thought "soft" produces a tendency to minimize symptoms which ought to receive medical attention. In any case a slight pain is a late symptom in the class of cases which we are considering, and it can be elicited only by special manipulations, according to the pathological type to which the particular case belongs—bimanual examination in slight endometritis with metritis, because the uterus is more or less sensitive to touch; deep lateral external pressure when there is a suspicion of slight perimetritis.

Is there foul-smelling lochial discharge? Frequently. The association of foul lochia and slight rise of temperature always implies the possibility of immediate danger, and it is characteristic of the only class which calls for manipulative treatment without delay. But, says an objector, "It is only a slight attack of sapræmia; it is not septicæmic." That is a very modern and entirely unproved, even unfounded, distinction. If you analyse the flood of verbal propositions which compose such a large part of the chapters on the pathology of the puerperium in so many of the text-books, you will find no clear exposition of a difference between sapræmia and septicæmia which can be promptly and effectively applied by the general practitioner. We are assured that the cases differentiate themselves in a few days, but a few days will make all the difference between satisfactory recovery and ruined health. I have my doubts as to "wait and see" being a satisfactory principle as applied either to the practice of midwifery or of politics. Foul-smelling discharge in child-bed demands active interference for the removal of the cause, not mere concealment of the odour by an antiseptic douche.

Now what are the *pathological processes* going on under our eyes, unobserved or undervalued, their import and possibilities seldom or ever exciting the interest of even the medical attendant? The process is a matter of *degree*, not of *kind*. It may be:—

- (1) Septic endometritis with metritis.
- (2) Perimetritis, strictly localized, in addition to the endometritis.

(3) Parametritis owing to lacerations, to such a slight extent as only to show its previous existence remotely by cicatrices and changes in the cervical mucosa.

It is not my intention to inflict upon you more than the average amount of platitude on prophylaxis, diagnosis, and treatment.

Prophylaxis is the application of all the practical knowledge we possess for the preventing septic conditions in child-bed. The greater includes the less. The medical practitioner does not allow his patients to suffer because of ignorance or apathy on his part. It may be admitted that he is often not so long-suffering as he ought to be, and that he readily conceives conscientious reasons for harmful interference. Then at the present transition stage he is frequently ill-served by half-trained, self-satisfied persons who have obtained a little superficial instruction in "monthly" nursing alone. So prevention is deplorably difficult.

As to diagnosis in such cases, it is possible to be too conscientious in the employment of manual methods. Efforts at exact diagnosis by manipulation may do more harm than good. Presumptive diagnosis with watchfulness is usually best for this class of patient.

The time for treatment, except in the more marked cases still within the category which we are discussing, has not yet arrived. The object of treatment is to prevent or minimize the consequences of the pathological processes, and the time for active interference is comparatively remote.

Let us take in more detail the pathological processes :—

(I) ENDOMETRITIS WITH METRITIS.

As soon as the endometrium becomes infected, the process of involution of the uterus is arrested. This is a fact which does not appear to receive the attention which it deserves. In the severer cases of septic infection which call urgently for treatment because of symptoms implying danger to the life of the patient, we often find, when we proceed to active treatment at the end of the first week of the puerperium, or even later, that the curette with a graduated stem will pass in for 6 in. to 8½ in. before it reaches the fundus uteri, and yet we are advised by some authorities to explore and clear out the cavity with our fingers! What digits they must be endowed with who can accomplish such a task! This arrest of involution is the chief cause of the pelvic condition observed remotely even in the slighter degrees of sepsis. In the typical case the patient leaves her bed at the time usual with the social class to which she belongs, and if she does not feel very robust, some of the

numerous minimizing explanations are found by neighbours or nurse, and accepted as sufficient. Sooner or later certain symptoms arise—the remote symptoms of the pelvic condition which must in the long run receive attention even from the most apathetic or patient of women. These symptoms are produced by conditions resulting primarily from subinvolution. They are usually a sense of weight or dragging.

On physical examination, say six months or more after the confinement, some displacement of the uterus will be discovered. The usual displacements are downward or backward, or both combined. If the examination has been delayed until the retroflexion has become chronic, it will be found that there is thickening with erosion of the posterior lip. If this condition is discovered, it considerably affects the prognosis as to cure by manipulations and pessaries. I have heard this anatomical change attributed to a local sepsis, and curettage recommended for its cure. This is, in my humble opinion, mere imbecility as pathology, and the coarsest and most irrational abuse of the curette, even in an epoch when curettage is the most outstanding and discreditable abuse in gynecological practice. I can remember a time when the guiding principle with many was: "If in doubt, introduce a pessary." Nowadays the principle appears to be, "If you don't know what is the matter, employ the curette."

If in a case of backward displacement of a subinvolted uterus the ovaries are dragged off their shelves, out of their fossæ, then cure without operation is not one of our experiences. Even in cases of uncomplicated retroversion or flexion, cure by manipulation and pessary is by no means common. One recent German writer puts the proportion of cure, in cases observed for from one to seventeen years, as 7 to 10 per cent. The largest proportion of cures amounts to 20 per cent. according to August Martin and Fehling. What becomes of the remaining 80 to 90 per cent.? They are condemned to "palliative" proceedings as a rule, and remain more or less under medical observation and treatment for the rest of their natural lives. This is the simple statement of a sad fact, which is perhaps not as a rule faced frankly and courageously by medical practitioners. During the child-bearing time of life sterility is by no means a consequence of these backward displacements without apparent cause. Impregnation, early symptoms of pregnancy and abortion form the usual cycle, occasionally varied by spontaneous rectification or retroflexion with incarceration of the gravid uterus.

(II) PERIMETRITIS.

When the sepsis in the slightest unobserved degree is conveyed by the lymphatics through the uterine wall or along the tubes, the result is peritonitis—the circumscribed peritonitis which we designate *perimetritis*. Even the very slightest peritonitis is followed, as direct effect, by some measure of adhesion; involution has been arrested or retarded early; the uterus is therefore large and heavy at the patient's getting up, and all the anatomical conditions favour the occurrence of complicated backward displacement. Whether tubes and ovaries become displaced or not, they at once take on certain processes of change which lead to important consequences. The tubes sooner or later have the fimbriated ends retracted and closed, or they become adherent to the ovaries; the ovaries gradually undergo pathological changes which may produce no specific and individual symptoms, but are fraught with important consequences. The tunica albuginea becomes thickened and sclerosed so that the Graafian follicles cannot rupture externally and shed their ova, but they must rupture within the ovarian stroma producing at first small cysts containing blood instead of forming corpora lutea. If such ovaries are not displaced they become adherent in their fossettes, clothed in the organizing peritonitic exudation which slowly but certainly contracts, doing still further injury to the ovary.

This process of organization of exudation and adhesions is by no means rapid; and for perhaps a year or so the bands are readily torn down. Later the adhesions may become as completely organized and injurious as those that result from puerperal perimetritis, obvious from the first because of the severity of the symptoms. They are separable only by cautious, prolonged manipulation, and they sometimes require the use of scissors.

TREATMENT.

(I) *Subinvolution with Displacement.*

The routine method of treatment has for its objects to diminish the congestion and bulk of the uterus, and to restore it to its normal position. These objects may be occasionally attained by medicated glycerine tampons and manipulations. The “medicated” is not essential; it is the glycerine which has the depleting effect. My favourite tampon consists of a long shred of lint, like a bandage, with 2 in. or so of the end

soaked in glycerine. The most useless of all tampons is the pledget of absorbent cotton which the patient is directed to apply herself. These methods are all purely palliative and temporary in their effects. The final resort is, as a rule, to the pessary, and the result is far from generally satisfactory. In simple hypertrophy with backward displacement the relief conferred may satisfy the patient and the doctor, but in the great majority of cases we condemn a young and otherwise healthy woman to discomfort and medical supervision for the rest of her life, for the menopause usually brings exacerbation. In retroflexion complicated with prolapse of the ovary nothing but harm can come from pessary treatment. It is astonishing how frequently the displacement of an ovary is overlooked in general practice, just as the complication of retroflexion with adhesions is not diagnosed in another category of cases; and the pessary treatment is persevered with in spite of the suffering resulting from pressure on sensitive parts.

In the long run, in most cases some sort of operation must be the last resort. In most of the old-standing cases with which we meet there is a history of curettage once at least. The theory, if any, at the foundation of this practice may be that once septic always septic, as may be seen seriously alleged by a contributor to a German gynæcological journal a few years ago. This on the analogy of Noeggerath's original doctrine that once infected with gonorrhœa, always gonorrhœal. I would suggest a method of treatment of the endometritis more gentle, as efficient, and without some of the serious objections to the curette, such as producing a wound which ultimately becomes a cicatrix. The uterine canal is cautiously dilated by means of a suitable laminaria tent, and a wick of gauze soaked in a solution of chloride of zinc is introduced up to the fundus, care being taken to neutralize the excess of the escharotic fluid. This application destroys a pellicle of the endometrium in a perfectly smooth symmetrical manner. The hypertrophy of the uterus is best reduced when there is some laceration of the cervix by an exaggerated Emmet operation, with the apex of the wound on each side reaching so high as to divide some branch of the uterine artery. Failing success of these minor measures, posterior colporrhaphy may be tried, here also by a rather exaggerated operation to make allowance for future shrinking; but, after an apparently successful operation of this kind, a pessary may have to be worn if the tendency to prolapse has been at all marked.

Among the operations suitable for uncomplicated prolapse with retroflexion is, I need hardly say, Alexander's operation of shortening the round ligaments. There is a distinct field of usefulness for the

original operation, which, it must be conceded, has a tendency to diminish in area. It fails to stand the test of pregnancy and parturition in about 20 per cent. of all cases.

PELVIC PERITONITIS.

By far the most interesting portion of the class of pelvic diseases concerning the origin of which we can obtain no clear evidence is that of complicated uterine and ovarian displacement, of which the cause or some part of the cause is pelvic peritonitis or perimetritis. Instead of a statement in general terms, an illustrative case will perhaps more clearly indicate the features of such a category. Such illustrative cases are by no means difficult to find. Here, *e.g.*, is a typical case of one-child sterility, from the practice of a man of wide experience and sound judgment in one of our most important Lancashire towns:—

Mrs. D., admitted to the Manchester Southern Hospital, October, 1899. The patient is aged 28; she has been married nine years, and has one child, born eight years ago. The labour was apparently normal and required no interference whatever. The patient got up at the end of twelve days, apparently well. She did not try to suckle the infant. In the course of a year or so Mrs. D. began to complain of certain discomforts, and Dr. —, after the usual examination, tried to replace the uterus, which he found retroflexed, by means of a pessary. This method was not successful, and the patient has been ailing to a slight extent ever since. There is no menstrual derangement; except within the last year or two the menstrual flow has gradually become more profuse and prolonged. It now continues seven days. At the hospital the case was diagnosed as retroflexion with adhesions. The patient was kept in bed for three weeks for treatment by douching and glycerine tampons, without appreciable benefit, so after due consideration by all interested the operation of ventro-fixation was performed. The uterus was found to be bound down by firm, but not extensive, adhesions. These were dealt with in the usual way, and the operation was completed. Patient was kept in bed for five weeks after the operation, although there was no incident to suggest special caution, and she went home perfectly well.

Let us supplement this case with another, in which the adhesions resulting from perimetritis were not allowed time to become firmly organized. It also illustrates the futility of some of our methods of treatment in complicated backward displacements, such as the Schultze process:—

Mrs. J., aged 28, with history of some slight illness after miscarriage during the first year of marriage. When admitted to the Manchester Southern Hospital in October, 1891, she was found to have retroflexion, with adhesion

of the uterus. The ovaries were not displaced as far as could be made out by ordinary examination. The employment of douching, tampons, and the Schultze manipulations failed to rectify the position of the uterus. Operation of ventro-fixation, October, 1891. Uterus found to be adherent to pelvic floor; broad ligament folded back, but ovaries not dragged from their fossettes, to which they adhere. Adhesions broken down readily, some hæmorrhage from torn adhesions. Ventro-fixation completed in the usual way; smooth recovery. Normal pregnancy labour and puerperium in following year.

Now, as such cases could be multiplied indefinitely from the notes of the last twenty years, and the practical conclusion from these two early cases are obvious, I forbear from the tedious process of further story-telling. I do not propose to analyse and criticize the vast number of ingenious methods of treatment, mostly surgical, which have been proposed and adopted more or less in Europe and America; I should only like to state my own conclusions that all these fancy operations have failed, with the exception of those which enable the operator to inspect the field of operation. Incomparably the best of these operations for backward displacement of the uterus with adhesions or abnormalities of position resulting from pathological conditions of ovaries and tubes is *ventro-fixation*—that is, *hysteropexis hypogastrica*, when properly performed—and no such operation is properly performed in which there is not complete abstention from interference with the round ligaments and the corpus uteri, except for a very short distance immediately above the isthmus. With the field of operation clearly in view, any necessary repairs of tubes or ovaries can be readily effected. It is seldom, if ever, justifiable to sacrifice either tube or ovary.

Without going into detail, I may state broadly the practical conclusions thrust upon us by the frequent occurrence of the pathological conditions which I have endeavoured to describe—viz., not preceded by any observations of puerperal sepsis in the puerperium:—

(1) Every woman should be carefully examined within six or eight weeks after her confinement.

(2) If subinvolution without complication is discovered, treatment should be at once begun, with the object of bringing the uterus to its normal condition.

(3) If uncomplicated retroflexion is diagnosed, the use of tampons, followed by the temporary wearing of a pessary, may possibly be successful.

(4) If tampons and pessary fail to restore the uterus to its normal condition and position, adhesions must be suspected, and efforts made to break them down by manipulations under anæsthesia.

(5) Failing success by manipulation, ventro-fixation, with the necessary modifications, is the only rational operation.

(6) In every case of one-child sterility with retroflexion, whatever the negative evidence, puerperal sepsis to some degree should be assumed as the cause, and ventro-fixation resorted to.

DISCUSSION.

Dr. HERBERT SPENCER did not think that there was much in the paper to discuss. Part of it consisted, in the words of the author, of not more than the average number of platitudes with which everyone would agree, and part of it of dogmatic statements with which he believed very few would agree. There was no mystery about hysteropexy or Sir William Sinclair's method of performing it. It was a useful operation in certain cases of retroflexion, but, as was shown by Dr. Russell Andrews and others, it did not always give good results, and was sometimes followed by disaster. He had himself known pain to follow the operation in cases performed by himself and others, and thought on the whole that the result of shortening the round ligaments by the abdomen was preferable to stitching up the uterus, although he had known pain also after that operation. He had operated by shortening the round ligaments in a case where conception occurred a few weeks after the operation, and the pregnancy and labour pursued a normal course and the subsequent health of the patient was excellent. He must protest against some of the statements in the paper. One of these said, "in contrast with private practice, it is alleged that morbidity is unknown in some of our lying-in hospitals." He asked Sir William Sinclair for the source of that statement, which he (the speaker) had never come across in any work he had ever read. Then, again, what was the meaning of "ovaries are dragged off their shelves"? Could Sir William Sinclair give any authority for the implication that normal ovaries are ever *on* shelves in the living body? Then, again, "no noteworthy new knowledge has been added to obstetric science and practice since the middle of the eighteenth century." He did not see the slightest use in discussing statements of this kind.

Dr. CHAMPNEYS said that Sir William Sinclair's preliminary remarks, apologizing for the character of this paper, almost disarmed criticism. He would only speak on one or two points. In the first place, he did not think that the author was correct in saying that it was the habit in London to treat cases of sapræmia lightly. He believed that all teachers taught that, at the onset, it was impossible to be sure that a case of puerperal pyrexia was nothing but sapræmia. The point was to make certain that nothing gross was left inside the uterus, and, in severe cases, to remove anything remaining. If symptoms ceased on providing for the thorough evacuation of the uterus, the case was one of sapræmia. The author had referred to the Central Midwives

Board (of which he was an original member), and to the great drawback from the survival of so many "*bona-fide*" midwives. It was not the fault of that Board that these women were on the Roll; but, indeed, it was an act of justice that they should be put upon the Roll, and this was in accordance with the practice of the Legislature in such cases. Now, this class of midwife doubtless included many who were highly undesirable, and such women were giving up practice, dying, and being eliminated by the Board at its penal sittings; but it also included women who were of great utility, and of whom local supervising authorities spoke highly. They had, it is true, to make bricks without much straw, but the bricks which they made were often uncommonly good. Sir William Sinclair spoke rather despondingly of the progress which was being made in the care of the poor mothers of the kingdom. In this respect it might interest the Section if he quoted from the Registrar-General's reports, which would be found in the Report of the Departmental Committee appointed by the Lord President of the Council to consider the working of the Midwives Act, 1902 (i, pp. 24 et seq.). From Table A, giving the annual death-rates from puerperal sepsis per million of females living, it would be seen that the death-rate in 1902 was 118, and in 1907 it was 81. The census of 1901 showed that in England and Wales there were 16,800,000 women. The saving of life in 1907, as compared with 1902, was 37 per million. In other words, the lives of more than 621 women were saved in 1907 which would have been lost in 1902. Table B, calculated in the proportion of 1,000 births, showed the same thing, and these results were graphically set forth in diagrams A and B. Diagram C, showing the death-rates from puerperal sepsis and accidents of childbirth to 1,000 births, showed that this rate prior to 1902 was never below 4'39; in 1907 it was 3'83. Striking evidence was given before the Committee to the same effect. As regards infantile mortality, Dr. Robinson, of Rotherham, stated that while the death-rate in cases attended by midwives was 101 per 1,000 in 1907, the death-rate in cases not attended by midwives was 194; in 1908 the mortality in midwives' cases was 92, in non-midwives' cases, 195. Dr. Champneys reminded the Section that the puerperal death-rate had not improved to any appreciable extent since Dr. Matthews Duncan's classical investigations published in 1871 until the present time. It was gratifying to find so substantial an improvement in the last few years.

Dr. MALINS said that he begged to traverse some of the remarks made by Sir William Sinclair as to the absence of any notable progress in our knowledge of midwifery during the past one hundred and fifty years, except, perhaps, that made by Semmelweis and Lister. The names of Edward Rigby, Denman, Simpson, and others, occurred to him as instances to the contrary. There was one direction especially in which great advance had been made during that period—namely, in our knowledge of the mechanism of parturition, as marked by the names of Saxtorph, Naegele, Litzmann, and others, to whom we are much indebted. With regard to the opinion expressed by Sir William

as to the present lack of intelligent appreciation of their duties by midwives and nurses, Dr. Malins said that the contrary was his direct experience, for that since the formation of the Central Midwives Board he had noticed, both from observation and experience as an examiner, a vast improvement in this respect. Dr. Champneys had given statistics in support of this, and, while the figures showed a great advance for so short a time, the facts pointed to the advantages gained and the good results obtained by the standard of knowledge required by this authority.

Dr. LEWERS said the author had spoken of cases of so-called "one-child sterility" associated with retroflexion. These he claimed to have cured by ventral fixation of the uterus. He had incidentally mentioned that in these cases separation of adhesions was necessary in order to restore the uterus to its normal position. Dr. Lewers thought that in the class of cases mentioned it was the adhesions, more or less completely occluding the fimbriated ends of the Fallopian tubes, that were the cause of the sterility rather than the retroflexion. He considered that if pregnancy followed the performance of ventral fixation in the circumstances mentioned, it was to be ascribed to the separation of the adhesions interfering with the patency of the Fallopian tubes rather than to the alteration in the position of the uterus itself.

Dr. EDEN said he understood the main point of the paper to be that a great many cases of chronic pelvic inflammation and of uterine displacement were due to slight septic infection during the puerperium which had not been recognized or treated. He was quite in agreement with this view, which had been impressed upon him by his experience of gynæcological out-patient practice. But with the explanation offered by Sir William Sinclair he could not entirely agree. So far from thinking that the distinction between sapræmia and septicæmia was a source of danger, he regarded it as of great practical usefulness. And, further, he thought that the work of Bumm with regard to septic endometritis in the puerperium marked a great advance in our knowledge, and justified the clinical distinction of the two varieties of uterine infection. Bumm's demonstration of the formation of a protective layer of leucocytic infiltration underneath the affected endometrium in putrid endometritis indicated the undesirability of curetting in the first week of the puerperium, for the destruction of this protective layer by the curette was an unjustifiable disturbance of the natural process of defence. The modern plan of avoiding curettage of the recently-infected placental site brought the treatment of the condition into line with the general surgical treatment of sloughing areas. Surgeons did not scrape such areas and so open up fresh channels for the absorption of infective material; they allowed the spontaneous separation of dead tissue to pursue its course, trusting to natural processes to isolate the area of infection. He thought these were sound reasons against the use of the curette.

The PRESIDENT (Dr. Macnaughton-Jones) said that with the general trend of the conclusions come to by Sir William Japp Sinclair as to the occurrence

of certain preventable complications arising out of labour he quite agreed, and it was a question if in all cases where such examination could be carried out, whether after natural labour or otherwise, an examination of the pelvic organs should not be made within a given time. But there were several matters in the paper with which he could not agree. He thought the criticisms on midwives were rather sweeping. Both as regards nurses and practitioners Sir William Japp Sinclair appeared to have had a very unfortunate experience. Surely no intelligent practitioner of the present day would attempt to empty a uterus, 8 in. long, with the finger. As to the terms "sapræmia" and "septicæmia," both from their derivations and the meanings ordinarily attached to them, he thought they should be kept totally distinct. Personally he always applied the term "sapræmia" to a local putrescence which generated toxins that were absorbed, and thus entering the blood, caused rapid and often fatal poisoning. "Septicæmia," on the other hand, he always associated with a general condition of blood infection, due to microbial or bacterial invasion. Then, again, he could not conceive that Sir William Japp Sinclair was speaking seriously when he said that the art of midwifery had not advanced since 1750. Since that date it had been elevated into a scientific art, worthy of the place it occupied in medicine generally. Since then it had been completely revolutionized. Take, for example, the changes in the blood in pregnancy, which were only first understood in the last century, and such important advances as those of Braxton Hicks and others. With regard to ventro-fixation, he understood from Sir William Sinclair that he always performed ventro-fixation, and not suspension. He quite thought that in a great number of cases the abdominal operation was preferable to Alexander Adam's, though he had always maintained that the latter operation had its own particular sphere of usefulness and was specially indicated in certain cases. But the abdominal method enabled us to explore the adnexa and deal with them if diseased, and thus save the woman from the ordeal of a second operation. He had performed suspension in a large number of cases by his own method; a kink of each round ligament being taken up, and the suturing of both done to the peritoneum and sub-peritoneal fascia at either side, a uterine suture also in some cases being added. He had never known of any bad results following the operation in labour. He maintained that, save in exceptional cases and for some special reasons where the adnexa had to be removed, ventro-fixation, with the fascia included in the suture, should not be performed during the child-bearing period of life. With regard to the allusion to the aphorism of Noeggerath, he (the President) thought it was meant to convey the fact that in some cases of gonorrhœal infection the effects were lasting and permanent. There could be little doubt of this latent existence of the gonorrhœal virus once it reached the adnexa and the grave consequences that frequently followed. They were indebted to Sir William Sinclair for having brought these practical matters before them for discussion, and for affording an opportunity for expression of opinion on the points that he had raised.

Tubal Mole, with Œdema of the Connective Tissue of the Villi.

By R. DRUMMOND MAXWELL, M.D.

CLINICAL history: C. H., aged 32, married; one child, aged 11; no miscarriages. There was a history of vaginal discharge and severe pelvic pain six years ago, when the patient was admitted to St. Bartholomew's Hospital. The diagnosis then made was pelvic perimetritis; the treatment carried out was expectant and palliative, and resulted in relief of symptoms.

History of present illness: Sudden onset of pain in hypogastrium, with vomiting on day of admission to Dr. Lewers's ward (London Hospital), March 20, 1910. The catamenia were regular till fourteen days before admission, when hæmorrhage started one week after the last period and continued slightly till the day of admission.

Physical signs on admission: A swelling is felt in the left ovarian region. *Per vaginam* there is a little blood-stained discharge. The uterus is fixed and approximated to the pubes. A very tender swelling extends across the posterior part of the pelvis. The temperature ranges from 99° F. to 102·4° F. The diagnosis was made of chronic tubal inflammation, with the reservation of tubal gestation.

Laparotomy, April 1, 1910 (Dr. Lewers): Much dark, clotted blood imperfectly encapsuled is seen in the pelvic cavity. Both tubes appeared at first sight to be the seat of ectopic gestation. Projecting from the ostium of the left tube is a dark blood-clot, the size of a pigeon's egg, which was spontaneously detached on manipulating the tube. The left tube was removed, sparing the corresponding ovary. Its infundibulum was expanded and dilated. The index finger was easily introduced through the dilated ostium, whence the mole had been extended. The right appendages on closer examination now appeared to present signs rather of chronic inflammation, though bathed in serum and blood effused from the opposite tube. The ovary contained numerous cysts of inflammatory nature; accordingly right salpingo-oöphorectomy was carried out. The ostium of the right tube is seen to be patent; on pressure a yellowish-brown fluid exudes through it, which is undoubtedly serum from the blood effusion occupying the pelvic cavity.

Many points of clinical and histological interest arise in connexion with the two tubes shown. Microscopic investigation of many cases of tubal gestation lends no weight to the view that chronic inflammation

of the tubes is at all a common causal factor; and, though in this case we had a clear clinical history and definite physical signs of a pelvic perimetritis due to an ascending infection, the power of regeneration of the tubal mucosa is well seen by the normal appearance of the epithelium in sections across the lumen of the two tubes. Both vesicular degeneration of a tubal mole and chorio-epithelioma primary in the tube have been described and recognized. Sections of the villi in this case, embedded in the wall of the right tube, have the typical structureless, bloated appearance of such pathological villi; and though there is no central cavitation in the villi, their structure, associated with



Microphotograph of case of tubal mole (obj. $\frac{1}{8}$). An œdematous villus occupies the bulk of the field. No trace of vascularization of its connective tissue is seen. To the right large masses of Langhans's cells are proliferating, and a few normal villi can be recognized.

the active proliferation of Langhans's layer, suggests that one is looking at a stage just short of vesicle formation. The proliferation of Langhans's layer is not, however, to be regarded as extreme in this situation, since in all abnormal sites of embedding of the ovum the epiblastic activity is always in excess of the normal when compared with the similar process taking place in the uterine decidua. This suggestion of cystic degeneration of the villi argues with some slight force against the expectant line of treatment so commonly successfully adopted in the less severe cases

of tubal gestation; since, had the symptoms and physical signs been less pronounced, the patient might easily have been left with a mole in the tube wall which seems to possess very suspicious features, and might possibly have given rise to a chorio-epithelioma of the tube. The difficulty of differential diagnosis between chronic tubal inflammatory disease and ectopic gestation is also illustrated by this case. True, the reservation of ectopic gestation was made in the diagnosis, but the history of perimetritis six years ago, the relative sterility of eleven years, the wide oscillation of temperature (99°F. to 102.4°F.), all favoured chronic inflammation of the tubes and, as usually is the case, there was no cast or shred in the discharge to assist diagnosis. One may perhaps look forward in the near future to a serum diagnosis of pregnancy as the only means of definitely settling this difficult differential diagnosis.

The changes in the opposite right tube are also interesting. Sections show a tube lumen which is absolutely normal, whatever may have been its condition six years ago. The main change is seen remote from the lumen, and is limited to the sub-peritoneal area of the tube. There is an acute inflammation here with free escape of blood into the sub-serosa. The change may very possibly be an expression of an early aseptic plastic peritonitis induced by the contiguous pelvic effusion of blood in a tube that has been at a remote date subject to catarrhal salpingitis.

Report of the Pathology Committee.—"We have seen the specimen and sections submitted by Dr. Maxwell under the title of 'Cystic Degeneration of a Tubal Mole,' and are of opinion that many of the villi show great enlargement, due to œdema of the cellular tissue. There is no actual cavitation of the villi, and we think the condition would be better described as 'molar tubal pregnancy showing extensive œdema of the connective tissue of the villi.'"

DISCUSSION.

Dr. LOCKYER agreed with Dr. Maxwell as to the extent of proliferation of the trophoblast seen in the section he exhibited of tubal pregnancy. Some of the chorionic villi were very much swollen by œdema, but the speaker was not convinced that the changes seen justified the diagnosis of vesicular disease of the chorion. Isolated areas of Langhans's cells and syncytial derivatives were to be observed, but such active proliferation of the trophoblast was not infrequently met with in tubal pregnancy, quite apart from any consideration of vesicular mole. Dr. Lockyer asked that the specimen be referred to the Pathology Committee.

Mr. ALBAN DORAN observed that repeated tubal pregnancy was known to be almost a common condition. He had reported a case in his own practice, in 1905, at a meeting of the Obstetrical Society. Since then he read notes of an operation at the end of the fifth month for extra-uterine gestation, with a living fœtus, before the Section, in December 1908. The pregnancy had developed in the left Fallopian tube. Last month (May, 1910) Dr. Eden operated on the same patient, removing a tubal sac from the right side of the uterus. The left ovary, Mr. Doran understood, was found to have undergone involution. These tubal pregnancies now so frequent, due, as Clarence Webster and others had shown, to decidual reaction extending from the endometrium to the tubal mucosa, possibly represented some general deterioration in the generative powers amongst civilized women.

Adenomyoma of the Fallopian Tube, with Tuberculous Salpingitis.

By J. INGLIS PARSONS, M.D., and BRYDEN GLENDINING, M.B.

THE patient, aged 33, had been married seven years and was sterile. She complained of dysmenorrhœa for many years. The pain was most marked on right side, just before and during menstruation, and prevented her from doing her work. In the intervals she had a general aching feeling in the pelvis. Menstruation was regular and not excessive. She suffered a good deal from constipation, and often had severe pain in the rectum during defæcation. The temperature was normal. On examination the uterus was found to be retroverted and held by adhesions, and tender to the touch. On the right side of the uterus a small, hard swelling could be distinctly made out.

Mr. Inglis Parsons opened the abdomen on April 12 by a median incision. The intestines and omentum were found adherent all over the anterior wall beneath the incision. After separation of these, both tubes and ovaries were found to be enlarged, and, together with the uterus and intestines, were matted together and held down by adhesions. On the right side a fibrous tumour, the size of a small hen's egg, occupied the Fallopian tube. All over the pelvis evidence of old tuberculous disease could be seen in the shape of small tubercles that had undergone either a fibrous regeneration or a caseous degeneration. Both appendages were removed, and all the adhesions separated. Two pints of saline fluid were poured into the abdomen. The patient made a good

recovery and her pains disappeared, while her general health improved very much.

Dr. Bryden Glendining's report on the specimen is as follows:—

“The specimen consists of: (1) A Fallopian tube, somewhat thickened with the abdominal ostium closed. On section the thickening is confined to the muscular wall. The cavity is not dilated. Microscopically there is a general fibrosis, with hyperplasia of the mesoblastic structures. The chronic inflammatory changes are marked in the subepithelial tissues of the villous folds and in the muscular coats. (2) A Fallopian tube and ovary. The ovary, rough and congested on the surface, appeared normal on section. The Fallopian tube is firm, closed at the abdominal ostium, and towards the uterine end is lost in a firm, spherical mass 3 cm. in diameter and covered by peritoneum. The Fallopian tube on section shows thickening of the wall. The mass, in the tube on section, shows a whorled appearance and, like a fibromyoma, tends to bulge out of the capsule. There are one or two white, soft areas the size of a pin's head. Microscopically in the Fallopian tube are areas of necrosis, collections of small round cells, and occasional giant-cell systems. These changes are confined to the muscular coats. In the subepithelial and in the muscular tissues there is chronic fibrosis. The mass in the Fallopian tube, microscopically, shows a fibromyomatous tissue, the fibrous and unstriated muscle running in strands and intersecting, in which occur gland spaces lined by columnar epithelium, and often adjacent to the glandular spaces, but sometimes distant from them, are areas of round-cell inflammation with giant-cell systems. Here and there are small areas of necrotic tissue; under the microscope the appearances are those of a tuberculous adenomyoma.”

Remarks.—There appear to be but few recorded cases of fibroma of the Fallopian tube, and none of adenomyoma. Dr. Lloyd Roberts showed a small fibroma of the right tube at Manchester.¹ Carrière and Legrand described a fibromyoma of the tube weighing 235 grm. (the sixteenth on record). The tube was normal and passed through the tumour.²

Report of Pathology Committee.—“We have examined the specimen submitted by Dr. Inglis Parsons under the title of ‘Tuberculous adenomyoma of the Fallopian Tube,’ and found that, continuous with the tube, and growing out of it, is an adenomyomatous tumour which, in common with other portions of the Fallopian tube, shows an inflammatory tuberculous process.”

¹ *Journ. of Obstet. and Gyn. of Brit. Emp.*, 1903, iii, p. 498.

² *Rev. de Gyn. et de Chir. abd.*, Par., 1902, vi, p. 437.

DISCUSSION.

The PRESIDENT asked what was the exact interpretation to be put on the term "tuberculous adenomyoma." It was not one that he had heard used.

Dr. BRYDEN GLENDINING said that since the term "tuberculous adenomyoma" had been questioned, and since he was in this instance responsible for that term, he would like to say a word in explanation. (1) In many inflammatory conditions of the Fallopian tube, especially in the chronic cases, there was produced an adenomatous condition of the endosalpinx which extended into the muscular wall to a considerable extent. In this case such a condition was present in the Fallopian tube and in the nodule of the specimen shown. (2) The tuberculous nature of the specimen was obvious. (3) The tumour histologically showed much fibrous and muscular tissue; the latter, much in excess of that normally present in the tube, also showed the typical whorled arrangement of a fibromyoma. The combination he called "tuberculous adenomyoma." He did not know whether such a term was recognized, but it described very well the pathological condition present.

Fibrosis of the Uterus causing Persistent Hæmorrhagia.

By J. INGLIS PARSONS, M.D.

THIS specimen was removed from a married woman, aged 48, sent to me by Dr. T. Jago. She has five children, and was first seen by me four years ago for menorrhagia. The uterus was then found to be enlarged. A diagnosis was made that the enlargement was due either to a general fibrosis or to several small fibromata. Hysterectomy was advised, because curetting generally fails to do any good in these cases. Operation was refused. The menorrhagia continued to increase gradually, and in October of last year had become continuous, while the condition of the patient was becoming serious from the constant bleeding. In December, 1909, the uterus was removed by me, a subtotal hysterectomy being performed. By clamping all the arteries first, before division, there was no loss of blood, and in spite of the very anæmic condition of the patient she made a good recovery and is now, Dr. Jago tells me, quite well and robust.

Dr. Eastes's report on the uterus is as follows: "The mucous membrane is fibrotic, being permeated by strands of new fibrous tissue containing thick-walled vessels. The musculature is thickened by hypertrophy of muscle, as much as, if not more so than, by a fresh

deposit of fibrous tissue. The latter is in excess, but is patchy and is not uniform. The vessels have their muscle coats hypertrophied and their adventitia thickened."

Report of the Pathology Committee.—"We have examined Dr. Inglis Parsons's specimen of 'Fibrosis of the Uterus,' and consider that both the naked-eye appearance and microscopic section confirm the pathological report submitted with the description of the case."

Double Uterus (Atresia of one-half), with Dysmenorrhœa.

By ARTHUR H. N. LEWERS, M.D.

THE patient, from whom the specimen shown was removed, was a single woman, aged 28. She had always suffered from considerable pain at the periods, since they began at the age of 14. This pain had increased in severity during the last four years previous to her admission into the London Hospital in September, 1909.

There was a history that an abdominal section had been performed in the provinces about three and a half years previously, and that the left Fallopian tube, closed and dilated with blood, had been removed. The dysmenorrhœa was not relieved by this operation, and subsequently the cervix was dilated on two occasions, also without any improvement resulting. As regards the dysmenorrhœa, the pain began the day before the period, and continued till a few days after the period was over. There were nausea and vomiting on the first day. The average duration of each period was six days, and the loss was moderate. As regards the physical signs, in September, 1909, the uterus was rather larger than normal, considering the patient was single; the increase in size was chiefly in width from side to side. On the left side there was a slight projection towards the peritoneal aspect about the size of a hen's egg. The opinion formed of this at the time was that it was probably a small fibroid. The sound passed a full 3 in. The cervix appeared normal. Shortly afterwards Dr. Lewers dilated the cervix again up to No. 14 Hegar, and the patient left the hospital with instructions to report as to the relief or otherwise of the symptoms.

She was readmitted into the London Hospital on November 10, 1909. She had had two periods since leaving the hospital, and the pain had been rather more severe than before the last dilatation. She was kept

in hospital over the next period for observation. This occurred at the beginning of December, and there was no doubt at all of the severity of the pain. Accordingly it was decided to perform abdominal hysterectomy.

Operation December 17, 1909: On opening the abdomen the sigmoid flexure and bladder were found to be firmly adherent to the stump left by the previous operation for the removal of the hæmato-salpinx. Careful dissection was necessary to separate the adherent parts. The right uterine appendages were normal. The uterus was seen to be enlarged in the transverse section, and during the operation the projecting portion to the left was believed to be a small fibroid. Supravaginal hysterectomy, with removal of the right uterine appendages, was performed in the usual way. The patient made an uneventful recovery, and left the hospital on January 11, 1910.

Examination of the specimen: On the left side of the uterus was a projecting mass measuring 2 in. by 2 in. by $1\frac{1}{2}$ in. On section the mass was found to have a central cavity measuring $\frac{3}{4}$ in. in diameter. This cavity did not communicate with the cervix. Microscopical examination showed that this cavity was lined by mucous membrane identical with that found in the right half of the uterus.

DISCUSSION.

Dr. GRIFFITH referred to a case of double uterus in a young lady suffering from severe dysmenorrhœa which was diagnosed only at an exploratory operation, and pointed out the great difficulty of diagnosis. He did not feel justified in removing any part of the uterus.

Dr. BLACKER thought the difficulty in coming to a diagnosis in these uncommon cases was well illustrated by a case on which he had operated recently. The condition was thought to be one of a dermoid cyst of the left ovary. On opening the abdomen he found that in reality he had to deal with a hæmatometra in the undeveloped horn of a unicornate uterus, together with a hæmatosalpinx and what appeared to be a peritubal hæmatocele. He hoped to bring the specimen before the Section at a future date.

Mr. ALBAN DORAN remarked that he had recently examined the preparation showing a hæmatometra in a uterus removed by Mr. Meredith, the case in fact to which Dr. W. S. A. Griffith had just referred. It was associated with a liquefying uterine fibroid, like Mr. Doran's own case of cystic interstitial uterine fibroid appearing papillomatous, reported in the *Journal of Obstetrics*, December, 1908. This condition was not necessarily painful, but appeared to be always accompanied by hæmorrhages. The hæmatometra in Mr. Meredith's case was no doubt secondary to the degenerative change in the uterine tumour.



